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Original Communications

THE DIGITAL MANIFESTATIONS OF SUBACUTE BACTERIAL ENDOCARDITIS

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IN any form of bacterial endocarditis the clinical course of the disease can be divided into two stages, a pre-embolic and an embolic stage. In acute bacterial endocarditis the disease commonly runs such a rapid course that the interval between the pre-embolic and embolic stages may be measured by hours. In the more prolonged forms of bacterial endocarditis, which have been described as subacute or chronic, the pre-embolic stage may occupy weeks or even months, and inasmuch as the recognition of embolic phenomena is of vital importance in diagnosis, even the slightest evidence of their presence is of value in such cases.

There are certain facts about the lodgment of emboli which are clinically important in the diagnosis of endocarditis. It is well known, of course, that there is a tremendous variation in different patients in the number of emboli which are detached from the diseased valves during the course of the process. It is also known that emboli are likely to occur in "showers" so that when embolic manifestations occur in one part of the body they are also likely to occur in others. It is further true that emboli, especially the smaller ones, tend to become arrested in terminal vessels and for this reason embolic manifestations are often prominent at the periphery of the body. It is the purpose of this article to point out the importance of some of the peripheral manifestations of embolism in subacute bacterial endocarditis and also of some other peripheral manifestations which are probably not embolic in origin but which are nevertheless of diagnostic value.

OSLER'S SIGN

Among the more common digital manifestations of subacute bacterial endocarditis are the painful nodules, which usually occur on the fingers or toes, to which William Osler called particular attention. It is quite

clear that Osler was not the first to recognize these nodules, for Heubner in 1899 gives a description of the finger lesions in one of his patients which leaves no doubt that he observed them. He describes the sign as pain in the finger pads and transitory swelling and reddening of the skin of localized parts of the finger, similar to erythema multiforme. Münzer in 1900 described a painful spot on the thumb of one of his patients. Rapin in 1903 describes what he calls "taches rosées" on the fingers and also speaks of painful, slightly indurated spots on the body "like flea bites." Osler describes the lesions as painful, ephemeral, erythematous nodules with a diameter of one-half to one and a half centimeters, red, papular, and often pale in the center, occurring mostly on the fingers and toes and generally disappearing at the end of some hours to a day. They appear most frequently on the pads of the distal phalanges near the nails (Murray). The lesions are never very abundant and generally occur in crops. As Parkes Weber noted they may be intracutaneous or subcutaneous, the former more frequently. As Nicholl has pointed out, the lesions occasionally suppurate and leave a small ulcer. More often they gradually change from a red to a bluish color and may leave a slight brownish stain behind them. Occasionally they leave a small scab which may be picked off.

Since Osler emphasized the occurrence of these lesions in 1908, their presence has been substantiated by numerous writers, particularly by Libman, but a survey of the literature shows a curious discrepancy in the frequency with which they have been observed in different places. For example, in a series of thirty-one cases from the Bulgarian Clinic at Cluj described by Calugareanu, no mention at all is made of Osler's nodes. Notwithstanding the fact that the lesion was first described by a German, Huebner, there is very much less about it in the German literature than there is in the French literature. French clinicians in recent years have frequently called attention to it and have named it "fausse panaris" or "panaris d'Osler," not a very happy description, as the lesions do not particularly resemble the ordinary run-around.

My personal experience in private practice and in the New Haven Hospital would lead me to believe that if the lesion is looked for it can be observed, or a definite history of it can be obtained, in approximately 40 per cent of all patients. It was noted in nineteen out of forty-eight personally observed cases. The lesion is not pathognomonic of the subacute form of bacterial endocarditis. I have seen it in at least one case of fulminating acute endocarditis due to the Staphylococcus aureus, but in the vast majority of instances the Osler lesion is suggestive of the subacute bacterial form of the disease.

TENDER FINGERS AND TOES

The Osler nodes represent lesions which have resulted from the lodgment of minute emboli in the superficial terminal vessels. Where em-

boli lodge in the deeper vessels of the fingers or toes the Osler nodes do not result. In the series of cases which I have observed there have been a number in which the patient has complained of localized pain and tenderness in one or more fingers or toes. It has been possible to demonstrate on examination of these patients that pressure upon the affected digit was painful, but it has not been possible to demonstrate swelling or redness. The assumption has been that these lesions were due to the lodgment of deep emboli and this has been substantiated in some instances at least by the simultaneous occurrence of obvious peripheral emboli. In one patient, for example, evidences of deep embolic lesions accompanied a partial blockage of the right femoral artery. These lesions are probably similar in nature to the tender toes which sometimes occur in connection with typhoid fever. It will be recalled that many years ago Lewis Conner suggested that these were of embolic origin.

SPLINTER HEMORRHAGES

In a series of forty-eight cases I have observed two in which a very interesting hemorrhagic lesion occurred under the nails. The name "splinter hemorrhage" is suggested for these lesions because it exactly describes them. The patient complains of a sore finger, just as is usually the case in connection with the development of an Osler node, and examination instead of showing the common Oslerian lesion shows a linear hemorrhage beneath the nail usually about 4 or 5 millimeters in length and several millimeters removed from the growing edge of the nail. These lesions so exactly simulate the appearance of a splinter that were it not for the fact that the patient is usually in bed and that the lesion is several millimeters from the growing nail edge, one would suspect that a splinter had been accidentally introduced.

I can find no description in the literature of these lesions although Horder speaks of "a vivid linear splash at the *side* of the bed of the finger nail" (italics mine). This description hardly corresponds to the lesions which I have observed. Somewhat similar lesions have been described in connection with erythema nodosum by Lendon and are called by him "Verco's sign." I judge from his description, "subungual striae or dots of hemorrhages," that these lesions do not closely simulate the appearance of splinters. It is possible that the "subungual ecchymoses" spoken of by Lereboullet and Mouzon may have been of this nature but their description does not make it possible to decide.

TROPHIC DISORDERS OF THE DIGITS

In the series of cases which the author has personally observed, there was one in which the terminal phalanges of the fingers were described as showing trophic disorders similar to those that are so often seen in connection with chronic arthritis and also in connection with hemi-

plegia and peripheral nerve disorders. These lesions affect most prominently the skin. This structure is markedly atrophic and has a characteristic white, shiny, smooth appearance quite different from the normal wrinkled skin. This single observation would indicate that this form of trophic change is of no particular importance in the diagnosis of subacute bacterial endocarditis.

CLUBBED FINGERS

It has generally been stated in medical literature that clubbing of the fingers is usually associated either with pulmonary lesions, particularly bronchiectasis, or else with congenital cardiac lesions. It is only of recent years that attention has been focused on the frequency with which clubbing of the fingers in various degrees is associated with the subacute form of bacterial endocarditis. As long ago as 1891, however, E. Bamberger observed such a case though he did not realize its significance. A glance through the literature of the disease shows that long before the condition was recognized to be of diagnostic importance, occasional histories of patients with this disease show its presence. In our series of cases we noted occasional patients with clubbed fingers as early as 1908, and among the forty-eight patients there were eighteen who had finger changes varying in degree from slight incurvation of the nails to typical Hippocratic fingers.

The English school of clinicians were probably the first to appreciate the frequency with which clubbing of the fingers occurs in subacute bacterial endocarditis. Cotton states that the frequency of the lesion was noted at the Sobraon Military Hospital in 1917 and in papers published in 1920 both he and Thomas Lewis emphasize the point. Among the French observers Gallivardin of Lyons noted in 1921 the importance of clubbing and Jungmann, a German observer (1921), states that since he knew of the sign he has seldom failed to find it. The most extensive discussion of the subject is by Cotton who shows that clubbing of the fingers in cardiac cases is associated with subacute bacterial endocarditis in about 70 per cent of the cases. He also shows that it is rather more apt to occur when the heart is enlarged, that the occurrence of cyanosis is not a necessary precursor, and that it may be associated with either mitral or aortic disease or a combination of the two. Antecedent rheumatism or syphilis seems to play little if any part in its development.

CONCLUSIONS

1. The detection of digital lesions in the form of Osler nodes, tender digits, splinter hemorrhages or clubbing of the fingers is of great value in the diagnosis of doubtful cases of subacute bacterial endocarditis.

2. No conclusions can be drawn at present as to the frequency of these different phenomena. The great discrepancies as to the frequency

of their occurrence which occur in the literature are probably due not so much to regional variations in their occurrence as to differences in observational capacity and to lack of knowledge of their occurrence and significance. It is probably safe to assume that as they become more widely known they will be more frequently observed and that the apparently great differences in their frequency according to different authors will disappear.

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A STUDY OF TWENTY-EIGHT CASES OF BUNDLE-BRANCH BLOCK

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WE have collected for study twenty-eight cases of bundle-branch block from our office and the Presbyterian and Polyclinic Hospitals. Occasionally but one examination of the patient has been made; other patients have been followed for months and even years.

In the group there were nineteen men and nine women. The youngest was a boy of fifteen years with congenital heart disease. Clinically he appeared to have moderate aortic atresia and a double mitral lesion. A septal perforation is problematic, as there was no postmortem examination. In the decades from twenty to forty years there were no patients; in that from forty to fifty years five; from fifty to sixty years six; from sixty to seventy years fifteen, and from seventy to eighty years one.

The diagnoses were chronic myocarditis, mostly with heart permanently enlarged, twenty-three patients; mitral disease with chronic myocarditis, four patients; aortic disease, one patient. Eight patients (six right branch, two left branch) had auricular fibrillation with their bundle-branch block. Some had it established when first seen, some developed it while under observation; in one patient it was paroxysmal. Most of them had a rather advanced degree of cardiovascular degeneration. The majority had reached the age when it is to be expected.

Attempting to trace any definite etiology in these patients is unsatisfactory, as the average hospital history is deficient in that it fails to record clearly the previous infections, especially rheumatic manifestations and chorea. The histories from the cardiac clinics are more satisfactory. As shown above, the majority of the patients are well past middle life and their recollections of their earlier medical history are hazy. There were positive Wassermanns in five cases only; only two gave a straight history of acute rheumatic fever; recurring tonsillitis occurred in two; severe influenza and erysipelas in one each. There were several histories in which the infections were passed over entirely. The marked complications were, nephritis in five cases, decided hypertension in four, cholecystitis in one.

The diagnosis of bundle-branch block can be made only by the electrocardiogram. The criteria we use in making this diagnosis are those given by Carter[1] and by Lewis[2]:

1. The QRS interval exceeds 0.1 second.
2. The QRS complexes show an increased amplitude.
3. The QRS shows notching in these exaggerated complexes.
4. T′ is large and opposite in direction to the main QRS deflection.
5. The main deflection of such a QRS complex directed upward in Lead I and downward in Lead III indicates a block of the right branch. The main deflection of such a QRS group directed downward in Lead I and upward in Lead III indicates a block in the left branch.

The former is primarily written by the left ventricle (levocardiogram), Fig. 1, the latter by the right ventricle (dextrocardiogram), Fig. 2.

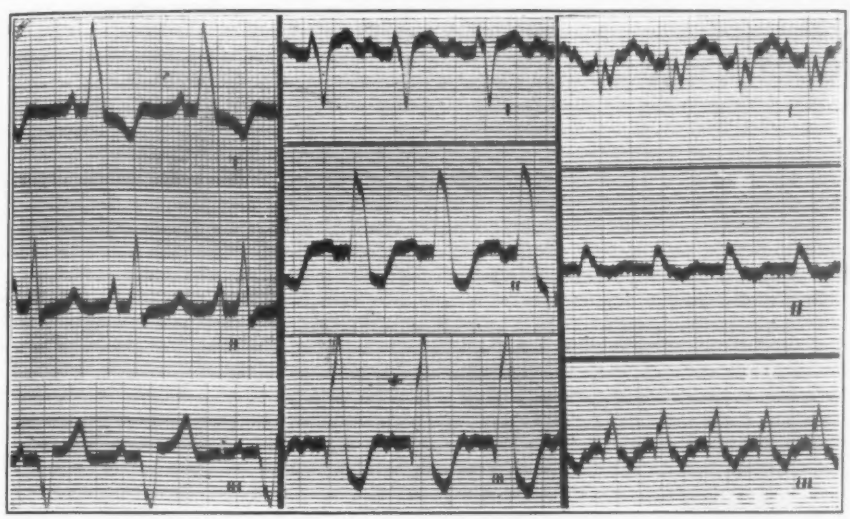

Fig. 1. Fig 2. Fig. 3.

Fig. 1.—Right bundle-branch block. Levocardiogram.
Fig. 2.—Left bundle-branch block. Dextrocardiogram.
Fig. 3.—Partial left bundle-branch block. Reduced voltage of a myocardium in advanced failure.

Our series includes twenty cases of right bundle-branch block, three of indubitable left branch block, one in which there was alternate temporary defect in right and left branches, and four suggesting partial block in the left branch, as illustrated in Fig. 3, taken from a patient who died two weeks later. All the criteria for diagnosis of left bundle-branch block are fulfilled in these four curves except amplitude. That this may be due to reduced voltage from a failing myocardium is suggested by the comparison of Figs. 4 and 5. Fig. 4, a curve of genuine left bundle-branch block, was taken Jan. 28, 1925; Fig. 5, from the same patient, was taken March 7, 1925, when the cardiac failure had markedly progressed. The patient, a man of fifty years with chronic

myocarditis, died of acute pulmonary edema March 9, 1925, two days after the last curve was taken. The resistance of the patient was taken with each lead, so they are technically correct. One would diagnose the first curve left branch block, but would hesitate over the second if it were the only curve of the patient one had.

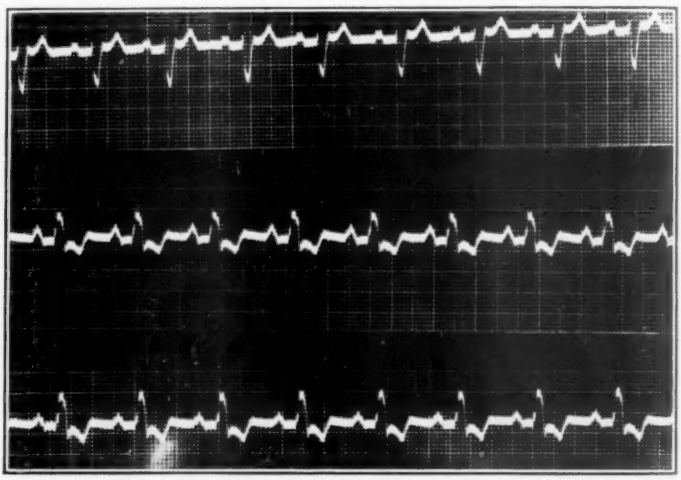

Fig. 4.—Left bundle-branch block. Taken January 28, 1925.

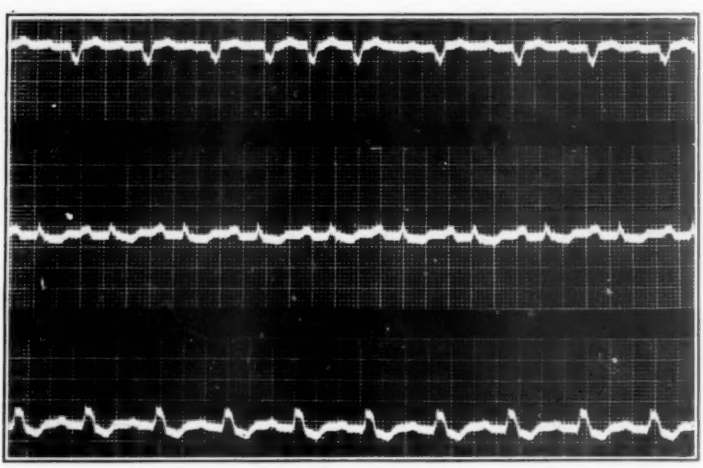

Fig. 5.—From same patient as Fig. 4. Left bundle-branch block with the low voltage of extreme failure.

That Fig. 5 is not due to digitalis is shown by the fact that the QRS complex is not more prolonged; it remains 0.12 seconds in both Figs. 4 and 5. Nor is the T′ deflection prolonged and increased in amplitude in Fig. 5 as compared to Fig. 4. In his cases of bundle-branch block Hart[3] found these changes in the T′, as well as a widening of the QRS complex under the influence of digitalis.

Partial block in a bundle leading to a shifting of the time relations of dextrocardiogram and levocardiogram and thus modifying the bicardiogram, as suggested by Wilson and Hermann,[4] is a most satisfying explanation of many similar curves. But in Fig. 5 it appears to be rather a matter of reduced voltage than any new change in the refractory period of the left bundle in the two curves. For these reasons four curves whose complexes fulfill the requirements for the diagnosis of left bundle-branch block, with the exception that their amplitude is somewhat lessened, are included. The advocates of arborization block may claim them. But the QRST amplitude is greater than the original curves called arborization block; furthermore, the T′ in our four curves (see Fig. 3) is of rather large amplitude and is of opposite

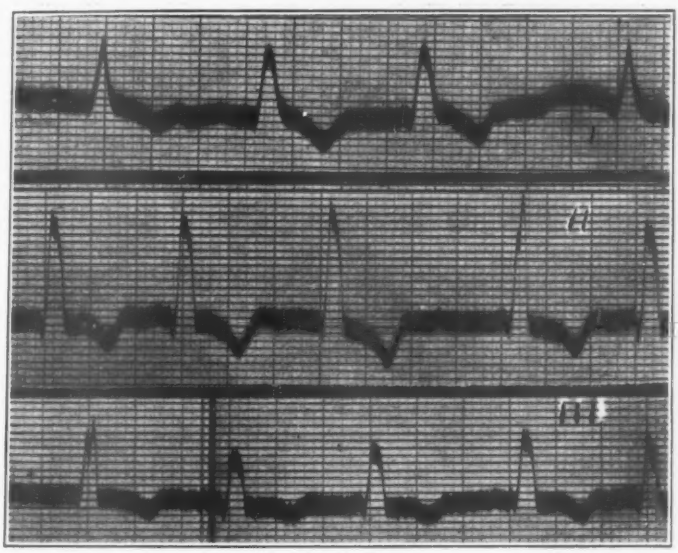

Fig. 6.—Left bundle-branch block. Concordant curve. Auricular fibrillation.

direction to S′ in Lead I and R′ in Lead III. The other three curves are much like those in Fig. 3. It may be more in keeping with our actual knowledge to call them simply intraventricular block. In any case they come nearer fulfilling the requirements for diagnosis of bundle-branch block than did several similar right branch curves which are not included. In these T′ usually failed in direction or amplitude or both, while the other criteria were perfect. We are aware that left branch defect is rare. This reminds us that once, in 100 consecutive patients in the office we found bundle-branch block (all right branch) five times,—an experience never repeated.

Fig. 6 we take to be a concordant curve of left bundle-branch block associated with auricular fibrillation. We have another curve where the regular discordant curve of right bundle-branch block was trans-

formed under digitalis into the concordant form. The patient was also fibrillating. In the latter curve there was the possibility that the leads might have been mixed. In Fig. 6 there is no such possibility. These curves probably simply illustrate some of the curious effects of digitalis on the intraventricular conduction.

The series included two patients with complete A-V dissociation; one had simultaneous blocking of the right branch, the other of the left branch. In but four patients was the P-R interval distinctly prolonged beyond one-fifth of a second. Five patients showed premature beats; two of these had right branch defect; one showed ventricular premature beats arising in the left ventricle, the other in both ventricles. One patient with right branch involvement and regular rhythm

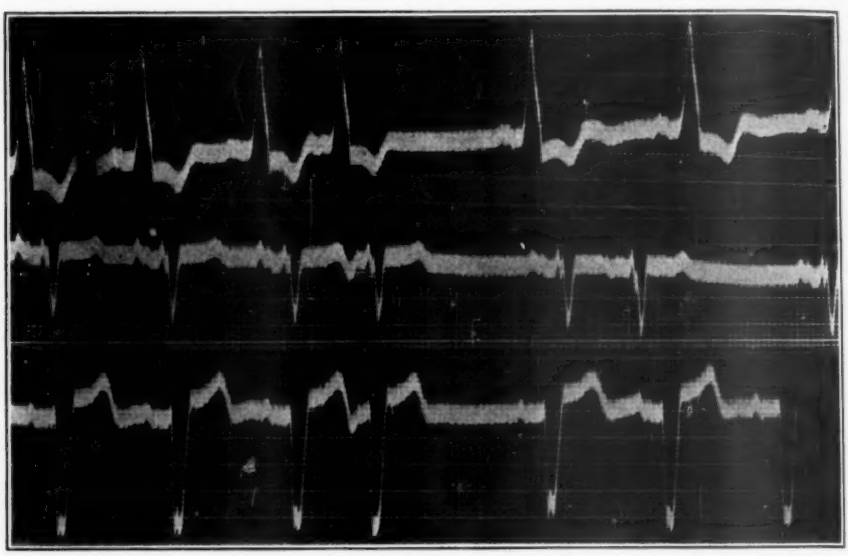

Fig. 7.—Right bundle-branch block. Auricular premature beats.

shows auricular premature beats. The P-wave is often inverted, but the ventricular complex is like those of the rhythmic beat (Fig. 7). One patient with left branch defect shows ventricular premature beats arising in the right ventricle, another shows them arising in the left ventricle.

In studying the curves one finds that Lead II was like Lead III in sixteen patients, (ten right branch and six left branch). Lead II was like Lead I in nine cases, (eight right branch and one left branch defect). Lead II differed from both Leads I and III in two patients.

In the patient with temporary right and left branch blocking, Lead II remained constant, while Lead I and Lead III changed their shape according to which branch was for the moment refractory. This patient will be reported fully later.

TREATMENT

Treatment does but little for these patients. Thyroxin, atropine, and adrenalin give no lasting benefit.

The syphilitic cases were so far advanced that antiluetic treatment had practically no effect. Occasionally in patients with beginning symptoms of congestive cardiac failure digitalis seemed to help, but the limit of tolerance was reduced, as many premature beats appeared early. Beyond this stage digitalis aggravated the condition. Complete rest in bed was the greatest help to those who reacted at all. Some of these patients were readmitted to the hospital several times.

PROGNOSIS

The outlook is bad for these patients. Of this series eighteen are dead; ten with right, seven with left branch involvement, and the one with both branches affected. Ten are living, all having right branch defect. Of the eighteen known dead, one woman lived three years after her block was discovered, and two women lived each one and one-half years. The other fifteen died in from three to six months. The interval was shorter for the left branch cases; only one survived as long as three months after the lesion was found. Age played little part in prognosis. The boy of fifteen years died six months after the diagnosis was made. Those between forty and fifty years, sometimes luetic, succumbed in two to three months.

Of the ten patients living with right branch involvement, six are men and four are women. Three were diagnosed only a few months ago. One man had lived four and one-half years, but we now know from recent normal curves that his blocking must have been temporary. One woman of whom we have several curves has right branch defect, found at sixty-seven years. She had her appendix removed at sixty-nine, and is living fairly comfortably at seventy-one, four and one-half years since her condition was found. She lives a sheltered life.

A man of sixty-three has gone three years, and four other patients one year each since the diagnosis was made. However, they are all always on the edge of cardiac failure with congestion, which is the commonest terminal stage. This was the usual end of the majority of the eighteen patients dead. Two died of acute pulmonary edema and one was found dead in bed.

SUMMARY

Twenty-eight cases of bundle-branch block are reported. They include nineteen with right branch block, one with temporary right, three with characteristic left, four with left, with reduced voltage, and one with both temporary right and left branch defect. All showed some degree of cardiovascular sclerosis, many an advanced degree.

Prolonged periods of rest averted for months or years the final breakdown. Restricted use of digitalis helped at times.

The outlook is poor. The majority die in a few months. The degree of cardiac degeneration is more important than age in prognosis. In this series the left branch cases were among the shortest lived.

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HEART-BLOCK WITH AND WITHOUT CONVULSIVE SYNCOPE*

Spectacular Therapeutic Results from Barium Chloride
Consideration of the Physiological Mechanisms Involved in the Effects of the Therapy and in Conduction Disturbance in General

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THE chemicophysical mechanism of the genesis and propagation of the excitatory process in the heart is at present the subject of the most intensive studies by cardiac physiologists. This fertile field has yielded and is still yielding results that are directly applicable to the cardiac problems of clinical medicine and therapeutics. The appreciation of the value of electrocardiography and the wide use of the method in the study and treatment of heart disease has been responsible for the recent rapid advances in our knowledge of the pathological physiology and physiological pharmacology or therapeutics of cardiac disturbance. The discovery of clinical cases that reproduce the experimental findings is usually at the heels of the announcement of the results of the investigation. At times the clinical report antedates, inspires and awaits the confirmation of the experimental studies. The cold-blooded heart, such as the turtle's has proved ideal for experimental work because of the facility with which investigations can be carried out for the initial studies and the revelation of the physiological laws which can be and usually are confirmed with much less complete corroborative evidence in the warm-blooded heart by later investigations. With the one possible exception of the apparent difference in the effect of vagus stimulation upon block in auricular muscle, increasing it in the turtle (Garrey) and decreasing it in the dog (Lewis), which after all may depend upon differences in the condition of the muscle, we know of no fundamental difference between the experimental results in the reptile heart and in that of the mammal.

In this paper we will present two clinical cases which are of unusual interest from the standpoint of the therapeutic results, as well as of the electrocardiographic findings. The close relationship between the pathological physiology, as revealed in the electrocardiograms, and the pharmacological reactions, and the therapeutic result, is very evident. We shall call attention to these points and suggest their bearing on the physiology of heart-block.

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The first case is one of unusual therapeutic interest. In the first place the Adams-Stokes attacks, and in fact all of the cardiac symptoms, seemed to have been precipitated by the potassium in the routine mixed antiluetic treatment. This regime was instituted on the patient's first visit to the out-patient clinic because of the evidence of cardiovascular lues. The possible ill effects of morphine and atropine, the reaction to epinephrin and the spectacular results of barium chloride are the outstanding things of clinical interest. The graphic registration of the effects of drugs, the effects of changes in the auricular rates, the shifting of the idioventricular pacemaker, the record-breaking prolongation of the A-V conduction time (the longest recorded P-R interval), and a supernormal phase are the outstanding electrocardiographic findings (Fig. 1).

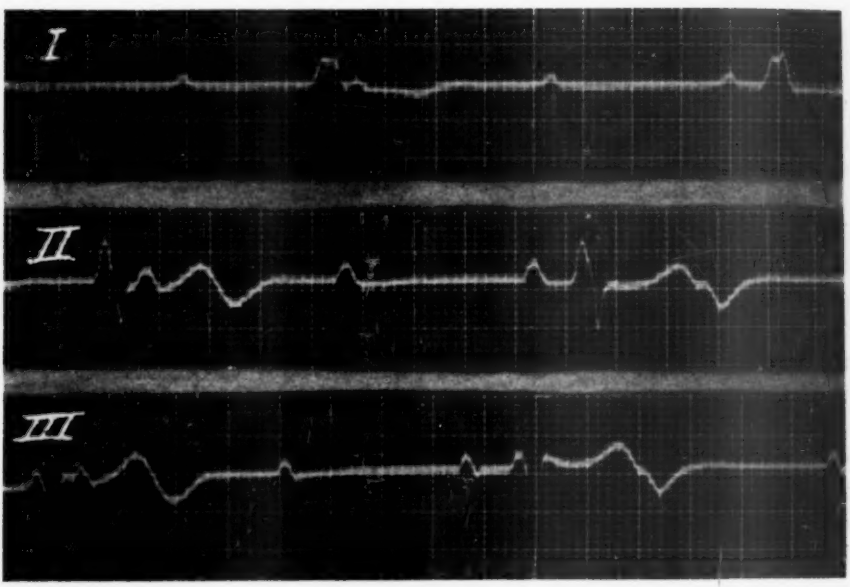

Fig. 1.—(Case I.) The three leads from above downward, I, II, III. First electrocardiogram taken, showing typical complete heart-block. The QRS group is broad, suggesting either that the idioventricular pacemaker is low in the left branch of the His bundle or that conduction through the specialized tissue of the ventricle is generally depressed. The patient had had barium chloride therapy for several days.

The second case, though improved, is not so striking from a therapeutic standpoint. The heart-block had apparently caused little trouble other than perhaps a slight dyspnea because of the slow rate and the lower minute-volume output. The blood pressure was high, part of which was probably a compensatory phenomenon. Drugs, such as barium, atropine, adrenalin and mixed antiluetic treatment produced no striking clinical results. The electrocardiographic studies, however, were most interesting. The effects of drugs and auricular rate changes on the degree of block, the active idioventricular pacemaker, the as-

sociated defective conduction in the right bundle branch, the striking combination complexes which reproduce previous experimental findings, make the case worthy of careful study (Fig. 2).

CASE HISTORIES

CASE I.—M. E. E., a white retail hardware salesman, aged forty-two years, was first seen by Dr. E. Z. Browne and later by Dr. G. R. Herrmann in the medical clinic of Charity Hospital, and with the onset of syncopal attacks he was sent into the ward of Professor John H. Musser for observation and care.

On admission to the clinic the patient's symptoms had been vague and indefinite. He had had a "cold" with a cough for three weeks and because of this he had gone to the state tuberculosis clinic for an examination of the lungs. No evidence of pulmonary disease was discovered, but he was not satisfied with the

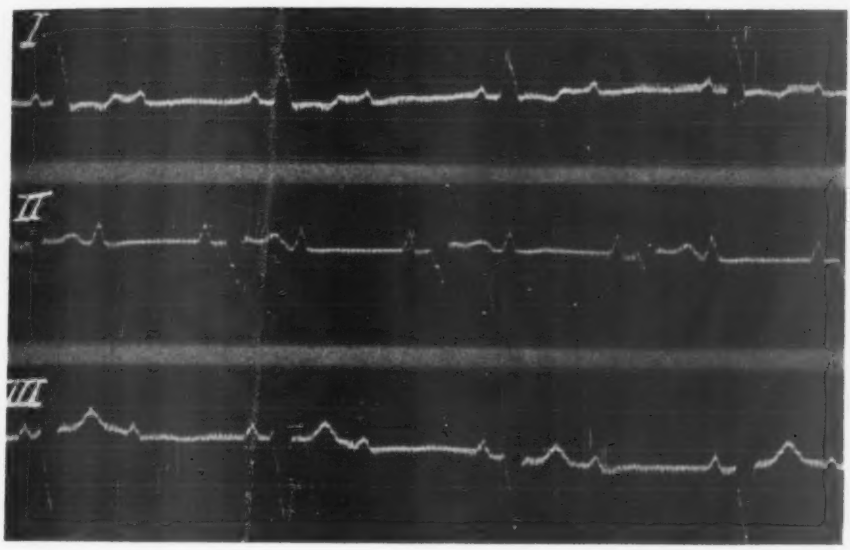

Fig. 2.—(Case II.) The three leads from above down, I, II, III. First electrocardiogram, showing a typical 2:1 A-V block together with defective conduction in the right branch of the His bundle.

examination. On close direct questioning in the clinic he admitted having had shortness of breath on exertion. He had, however, never had any swelling of the feet, hemoptysis or pain in the precordium or the right upper abdomen. He had not had his usual enthusiasm and energy for a year.

In his *past history* he had had pneumonia and typhoid fever in childhood, but no tonsillitis, chorea or rheumatic fever. He had had occasional attacks of "grippe"; malaria at thirty-five; gonorrhea, a right bubo and a probable neisserian arthritis at twenty-eight years. Syphilis by symptoms and by name was denied. The patient had never used alcohol, but had always used tobacco moderately.

In his *family history* it was noted that his mother had died of a stroke, his father of cancer and one brother and one sister of tuberculosis.

His *marital history* was of negative interest. His first wife had died of heart disease of a low grade infectious nature, possibly a subacute bacterial endocarditis. One son from this wife was living and well at the age of thirteen years. There

had been no miscarriages. His second wife was living and well and had had two normal children who were living and well at the ages of seven and three years.

Physical Examination revealed an aortic facies, a conspicuous pallor, throbbing of the carotids and nodding of the head. The pupils were round and equal and reacted actively to light and in accommodation. The teeth were in poor condition; there were many carious ones, many gold crowns and pyorrhea. The tonsils were not abnormal and no cervical adenitis was present.

The chest was slightly funnel-shaped. The lungs were clear and resonant throughout.

The *heart* was enlarged, the apex impulse was in the fifth interspace 14 cm. to the left of the midsternal line. No thrills or shocks were felt. A loud to and fro aortic diastolic and systolic, more or less continuous murmur was heard in the second right and in the third left interspaces. The aortic diastolic murmur was transmitted to the apex and its character was changed to a slightly lower pitch. A systolic murmur was also heard in the mitral area and transmitted toward the left axilla. No gallop rhythm was noted. The cardiac rhythm was regular and the only recorded rate was 64 per minute.

A conspicuous throbbing of all the peripheral vessels and of the finger tips was noted. A capillary pulse was elicited. A pistol shot sound and a Duroziez's diastolic murmur were heard in the femoral artery compressed by the bell of the stethoscope. The blood pressure was 168/40 mm. in the left and 150/50 in the right brachial artery.

The abdomen was negative. The liver and spleen were not palpable. The extremities were negative. No edema was present. The reflexes were active and equal. There was no swaying in the Romberg position.

The urine was negative. The blood showed a slight secondary anemia. The complement-fixation test was negative.

The x-ray of the heart showed an enlargement. The cardiac shadow measured 17.3 cm. in diameter. The aortic hemicircle measured 4.9 cm. across. The hilar lung shadows were exaggerated.

Treatment and Course.—The patient was put on potassium iodide in saturated solution, ten drops after each meal and mercurial ointment rubs each evening for six days of each week. Within fifteen days he noted an increase in his shortness of breath, a tired feeling and an actual weakness. He continued his treatment regime and on the thirtieth day he began to have sudden severe weak spells with a fainting sensation and dizziness. The heart rate was noted to be 28 per minute; two distant, slightly muffled auricular beats were heard in the long diastolic periods and a changing of the character of the first sound at the apex was noted and the patient was sent into the hospital as a case of complete A-V block. It was learned that on the day preceding the vertigo attacks the patient had exerted himself unduly by scrubbing the floor and washing the windows in a house into which he was about to move his household.

In the hospital the patient's pulse ranged between 28 and 54 per minute, remaining for the most part at the lower rates. *Barium chloride* was ordered for him, in doses of 20 mg. per os, every four hours. The interne failed to notice that the dose was ordered in the metric units and gave 20 grains; that is, approximately sixty times the dosage. Aside from nausea and vomiting and a slight diarrhea which was augmented by magnesium sulphate, there were no untoward symptoms. The slow absorption of the drug probably accounts for the absence of toxic effects. The dosage of 20 mg. every four hours of barium chloride was resumed and continued. Calcium lactate was given in 4 gm. doses along with the barium chloride. Tincture of digitalis was ordered in 20 minim doses t. i. d., but the patient received apparently only 2 c.c. in all. The patient fell twice during fainting attacks in the

hospital. He left the hospital after two days but took the barium chloride powders with him and continued the dosage of 20 mg. every four hours.

In the afternoon of the day on which he went home, he fell forward to the floor from the chair in which he had been sitting. This attack of faintness was similar to the two that he had had in the hospital. Recovery of consciousness was prompt. At this point an outside physician was called and morphine was given as a cardiac sedative. Shortly after the morphine was administered the patient had a much more severe "spell," unconsciousness with some convulsive movements and a pulse rate of 16 per minute—a typical Morgagni-Burnett, Adams-Stokes attack. He slept after the attack and six hours later, that is, at midnight, he had a still worse "spell," in which he was unconscious for fifteen minutes, during which his eyes rolled upward and set. Twitching of the muscles, profuse perspiration and a drop in the pulse rate to 8 per minute occurred.

A dose of .7 gm. (10 grains) of calcium lactate was given along with hot coffee and brandy stimulants. The calcium lactate seemed to cause gastric irritation, with nausea and vomiting, and was therefore discontinued.

The following day slight attacks were noted, even though the pulse rate had risen to between 26 and 34 per minute. Another outside physician was called and he advised and administered atropine. This drug also seemed to precipitate trouble, for shortly after its administration a long series of frequent and fairly long severe syncopal and convulsive attacks began. At this point one of us was called in and advised a doubling of the dosage of barium chloride (which had been continued at 20 mg. every four hours day and night), to 40 mg. every four hours, and the use of epinephrin (0.5 c.c. of 1/1000 sol.) intramuscularly and if necessary intracardiacly in the severe attacks. After the third dose of 40 mg. of barium chloride, a distinct improvement was noted. Attempts to defecate and empty an overloaded bowel precipitated the last syncopal convulsive attack that the patient has experienced. The dosage of the barium chloride was increased to 50 mg. every four hours for six doses per day and continued at this level for a week. The pulse gradually rose to 34 and at this point electrocardiograms were taken. Then the patient was advised to take only three 50 mg. doses per day and he continued to do this for eight weeks. The pulse gradually rose to 46, 54, 64 and finally 72. At this level the patient was advised to take but one dose of 50 mg. per day and he is continuing at this level. He has been back at his work as salesman for ten weeks; that is, he returned to his work within two weeks after his last attack. He is also receiving bismuth salicylate 0.13 gm. (2 gr.) intramuscularly every five days, as an antiluetic measure. He continues to feel quite well.

CASE II.—B. M., a negro farmer, aged 58 years, was first seen and his condition diagnosed by Dr. Hull W. Butler in the medical clinic of Charity Hospital and sent into the ward of Dr. S. Chaillé Jamison for further study and treatment.

He complained of weakness and shortness of breath. His symptoms had been present for about fifteen months. At the onset, while riding a horse, he suddenly lost all use of his right side. He did not fall from his horse, but managed to get to his home. His right arm was totally paralyzed but his right leg only partially so. His speech was impaired. A gradual recovery was noted after the fourth day, but three weeks elapsed before the use of the right arm began to return. The paralysis disappeared entirely within a few months, but a general weakness, especially of the right side, persisted. The weakness was becoming more and more severe.

Shortness of breath had been noticed for a year on sudden exertion or after prolonged heavy muscular effort, but never in paroxysms or at night. There had been a slight respiratory distress. No cough, bloody expectoration, precordial pain, palpitation, edema or fever have been noted. He had lost some weight, dropping

from 142 to 133 pounds and as low as 130 pounds, during his present illness. Nocturia had troubled him a little for some years.

In his *past history* he had had mumps at the age of 25 years; smallpox at 30; measles at 35; influenza at 45; and gonorrhea at 52. He has used tobacco and alcohol excessively. He had sustained deep cuts on his left arm and leg when he was caught in a cotton gin. A paralytic stroke had ushered in his present illness.

His *family history* was relevant in that his father had died of heart failure at 65.

The *marital history* is significant in that his first wife had died of unknown cause after having five stillbirths and five children, three of whom died during the first year of life of unknown causes and only two of whom are still living. He had had no children by his second wife.

Physical Examination showed a well-preserved negro, not acutely ill or distressed and able to assume any position and walk about with ease. He was 65 inches tall and weighed 135 pounds. His temperature was 98.4° F., his respirations 15 per minute, at times of a Cheyne-Stokes type, and his pulse unusually slow, 34 to 38 per minute, at times regular and at times irregular.

The eyes showed an arcus senilis. The pupils were equal, symmetrical, regular, round and reacted promptly to light and in accommodation. The teeth were carious and the gum infected with pyorrhea and swollen.

The neck veins were slightly engorged and showed an extra wave between the regular groupings of venous waves at times. The chest circumference was 31½ inches (80 cm.). The respirations were chiefly abdominal. The lungs were negative, except for slight emphysema.

The *heart* was somewhat enlarged with the apex impulse in the sixth interspace 10 cm. to the left of the midsternal line. The cardiac dulness extended 11 cm. to the left and 4 cm. to the right. The retromanubrial dulness was increased. The aortic second sound was accentuated and of a somewhat musical quality. The mitral first sound was somewhat prolonged and slightly blurred. No murmurs were heard. A faint muffled sound, apparently that of an auricular systole, was heard in each diastolic period. The rhythm was regular at times and at times irregular. The rate was persistently slow, 34 to 38 per minute, and would not rise conspicuously, such as doubling, on exercise. Exercise made the rhythm more regular. The blood pressure varied from 210/90 to 150/80 mm.

The abdomen was negative except for the fact that the liver border was just palpable, hard and not tender. The genitals were negative; no scar was found on the penis and no urethral discharge was present. The prostate was slightly enlarged and boggy.

The extremities were negative. The reflexes were more active on the right side. There were no evidences of residual paralyses. The peripheral vessels were thickened, tortuous and calcareous in places.

The urine was negative. The blood studies were negative. The complement-fixation and precipitin blood tests were repeatedly negative.

The x-ray film of the heart showed a shadow that measured 16.3 cm. in transverse and 17.3 cm. in longitudinal diameter. The aortic hemicircle measured 5 cm. These findings indicate enlargement of the heart and dilatation of the aorta.

Treatment and Course.—While in the hospital the patient was first given six 40 mg. doses of barium chloride in a period of 48 hours, without any effect. He was then given 0.7 mg. (1/75 gr.) of atropine for five doses at four-hour intervals for a day and a half without effect. Then barium chloride was given again in 40 mg. doses three times a day for five days without effect. One injection of bismuth salicylate 0.13 gm. (2 gr.) was given. The patient was discharged from the hospital in about the condition that he entered.

The patient returned to the clinic, where Dr. Hull W. Butler again saw him and on the strength of his marital history and his color started him in on a course of mixed iodide and mercury treatment by mouth. The patient has improved remarkably subjectively after six weeks of treatment, has gained fifteen pounds, and his heart rate has risen to between 50 and 70 per minute.

THERAPEUTIC DISCUSSION

Barium chloride was introduced into clinical medicine by Cohn and Levine,[1] for the prevention of the recurrence of syncopal and convulsive attacks that accompany ventricular standstill and heart-block. In their series of three cases the drug accomplished its purpose spectacularly while it was administered regularly. Two of their cases died ten and seven months, respectively, following discharge from the hospital and from the cardiologists' care. The other case was at work a year after the course of treatment. These investigators seemed to prefer a short course treatment with the barium chloride, the reason for which is not evident to us. The first patient who died had received only 240 mg. of the drug in six doses within 48 hours and was given no more after this. After discharge from the hospital, the attacks began to recur and calcium lactate in 1 gm. doses was given three times a day. but sudden death occurred ten months after the course of barium chloride. There are no reports as yet on the use of strontium salts and the relative values of the members of the alkaline earth group. The other patient that died had received while in the hospital a course of 810 mg. of barium chloride (30 mg. doses thrice daily for nine days). After discharge the drug was continued in similar dosage for ten days, at which time a faith healer persuaded the patient to stop the drug. Within ten days severe attacks recurred necessitating readmittance to the hospital, and she was kept alive by epinephrin injections for about twenty days, after which barium chloride, 30 mg. four times per day, was instituted and continued regularly for 44 days, omitted for 36 days and resumed again for seven days after which the patient took the drug irregularly and also took thyroid tablets in doses of .06 gm. (1 gr.) three times daily. She died suddenly seven months after her initial treatment; that is, about three weeks after the discontinuation of the regular barium chloride therapy. The patient who was at work with complete heart-block one year after a single course of 660 mg. of barium chloride (30 mg. four times daily for four days, then 15 mg. four times daily for three days and none afterward) was more fortunate and is alive, we are inclined to believe, in spite of the discontinuance of the drug rather than because of it. We believe that the idea of a course or an intermittent use of barium chloride is likely to prove dangerous, principally because the patient is apt to become careless and neglect his treatment; while if he is impressed with the fact that his life depends upon the presence of a constant minimum concentration of the drug in his heart muscle to keep his idioventricular pacemaker irritable,

he will cooperate more conscientiously. As will be noted from our case history report, we have used regular, heavy dosage over a long period of time without any evident detrimental effects.

The use of barium chloride to increase the irritability of the ventricular specialized tissue so that initiating stimuli arise promptly and prevent any periods of prolonged ventricular standstill, was suggested by the results of animal experiments. In these studies Rothberger and Winterberg[2] showed that barium and calcium tended to increase the irritability of the ventricles as shown by the occurrence of ventricular extrasystoles, at first singly, and then in shorter and longer runs of tachycardia. Van Egmond[3] demonstrated that barium chloride exerts the same influence even after complete traumatic auriculoventricular block. Junkmann[4] has recently shown that barium chloride, while causing the onset and continuance of a rapid idioventricular rhythm nevertheless produces simultaneously an extreme depression both in excitability and contractility. The absolute refractory period of the ventricular muscle of the frog was tremendously prolonged.

Epinephrin or adrenalin, however, still remains our drug of choice in the acute emergency of ventricular standstill, under which circumstances it is given in 0.5 c.c. dosage directly into the heart or into the jugular vein or even intramuscularly. The intramuscular and subcutaneous methods of administration are of doubtful value in real emergencies, for to be effective the drug must get to the heart, which is, of course, impossible from the last two routes when there is circulatory standstill. The effect of the drug is unfortunately very transient, lasting for an hour at the very longest. Furthermore, there seems to be no tendency toward the prevention of recurrence of the attack after the administration of the epinephrin, and it would therefore necessitate an altogether too frequent repetition of the injection to insure against ventricular asystole. We have used epinephrin in syncopal attacks with good results for the past five years. Phear and Parkinson[5] summarized the experimental and clinical literature on the effect of epinephrin on the heart and reported favorable results with the use of the drug in a human case of Adams-Stokes' disease. These authors abolished the seizures but produced no decrease of the block. They, however, cite two reports of cases in which actual disappearance of block occurred and four cases in which there was no change in the degree and character of the block. Experimental work showing unblocking, and failure to confirm these experimental results by two other groups of investigators are quoted. Two cases of aggravation of syncopal seizures and ventricular pauses are mentioned.

Korns and Christie[6] report an actual increase in the degree of auriculoventricular block in a case as the result of the injection of epinephrin. These authors rule out the possibility of peripheral vagus effects on the fact that full atropinization was without effect, but consider a

central vagus effect probable because epinephrin, which is known to stimulate the vagus strongly, causes no block if the vagi are cut. It seems to us that the block-increasing effect of increased auricular rate, an effect not considered by them, is the simpler explanation of their interesting observations. Epinephrin accelerates the auricles, often irregularly, and the ventricles, and produces frequent ectopic beats according to both these authors and Meek and Eyster,[7] through its direct action on the accelerator endings. Garrey[8] has shown that the accelerator stimulation increases conductivity and may relieve block. This fact together with the possibility of injury to vagus endings, with resulting reduction in tone, in the same process that is producing the conduction disturbance, would in our opinion explain the mechanism in the cases where unblocking results. It is merely a question of which action is predominant, good vagus tone and auricular rate increase or accelerator effects and poor vagus tone. We believe that the instances of exaggeration of syncope and ventricular pause produced by epinephrin in cases of complete block are the result of transient ventricular fibrillation.

The paradoxical effects of *atropine* in cases of heart-block must likewise be explained. Usually, and this is always the case where vagus tone plays any great part in the block, atropine through paralysis of the vagus endings abolishes the block and proves to be a very effective therapeutic agent. In rarer cases, however, such as Cohn and Levine's Case I and our Case I, in which atropine not only was without effect but actually aggravated the symptoms, we are inclined to believe that, through prolongation of the refractory period of auricular muscle or, more commonly, increase in the auricular rate, there is an increase in the degree of block out of proportion to the improvement in conductivity that is caused by the release from vagus influence. In *complete* organic (nonvagus or nonfunctional) block atropine of course has no effect.

The effects of digitalis and probably of morphine also, from the increase in vagus tone, might prove disadvantageous in cases of partial block by increasing the degree of block even to the production of complete block.

In our own cases the facts brought out in the above discussion are illustrated. In the first place, however, our first patient's severe symptoms seemed to have been precipitated by the mixed antiluetic treatment that he had received for about thirty days after his first appearance in the clinic. We are inclined to attribute these disturbances to the depressing effects of potassium salts producing or increasing an already present conduction disturbance. In the second case there was not only no such effect from the mixed treatment, but actually an increase in conductivity, a decrease in the degree of block during the course of treatment, as will be described more fully later.

The apparently detrimental effects of the morphine in Case I may possibly have been associated with increase in vagus tone, but we are not satisfied with this explanation. Tincture of digitalis, a total of 2+ c.c. had been administered in the hospital over a period of two days and no exaggeration of symptoms was recognized at the time. However, the first two attacks in which the patient was temporarily unconscious and fell, occurred during this period. This may have been a vagotonic effect of digitalis. The harmful effects of atropine in this same patient, we consider due to an increase in auricular rate out of proportion to the release of the vagus tone in the conduction tissues, and this increased the degree of block. We find evidence in the later electrocardiographic studies supporting these contentions. The barium chloride certainly increased the irritability of the idioventricular pacemaker and seemed in Case I to initiate, or at least to be accompanied by, a shift in the position of the pacemaker to a higher and higher level in the specialized tissues of the ventricle as suggested by the electrocardiograms. The idioventricular focus was so irritable that the higher degrees of partial block could not be produced because of the interruption of even a short quiescent period by a stimulus from the ventricular center. The patient has shown a definite but slight gradual improvement in conduction in spite of, perhaps rather than because of, the continued barium chloride therapy.

A CONSIDERATION OF THE MECHANISM OF HEART-BLOCK

Cases such as these two present many phenomena the significance of which would be much clearer if a detailed explanation of the mechanism of organic heart-block in its various manifestations were available. In view of the large mass of experimental work published within the last decade, it now seems possible to attempt such an explanation. But before offering our interpretation, it is necessary to review briefly a few of the theories hitherto proposed. Such conceptions as those of Straub[9] (after Hering), and more recently of de Boer,[10] which regard the refractory period of the ventricular muscle proper as an important factor in heart-block will not be discussed. Modern opinion is practically unanimous in the belief that, with possible rare exceptions, the propagated disturbance or impulse which fails to elicit a ventricular response, fails because it does not reach the muscle in question. The impulse is extinguished in supraventricular conducting tissue in which conductivity is impaired.

The first detailed theory of heart-block was that of Erlanger.[11] This author interpreted heart-block on the basis of two chief factors: the *intensity* of the auricular impulse delivered to the ventricle, which he believed decreased with increase in the degree of injury in the conducting tissue, and the *excitability* of the ventricular muscle. Facts not known at the time his ideas were advanced have since made questionable

certain of his assumptions. The most important of these facts is the demonstration that an impulse which succeeds in passing a region of depressed conductivity and reenters normal muscle, immediately regains its normal intensity and velocity (Drury[12]). Garrey[13] went much further and insisted upon the importance of factors not taken into account by Erlanger. Garrey gave reasons for believing that the refractory period of the injured muscle, as well as the intensity of the impulse arriving at that tissue; the excitability of the tissue beyond the injury, and fatigue were important factors. His paper implied strongly, if it did not explicitly state, that the condition of the ventricular muscle proper was not a factor of importance in A-V block, since the blocked impulse did not reach that muscle. Lewis and also Drury have recently made valuable contributions to our knowledge of conduction in the heart, and have done much toward clarifying our conceptions on the subjects. Their work will be referred to below.

The experiments of these and of other investigators indicate that any adequate interpretation of heart-block must take into account the nature of the impulse, its mode of transmission, its intensity, and the responsiveness of the injured tissues as these are influenced by changes in heart rate, by fatigue, or by other conditions. We shall therefore discuss these factors and then develop an explanation of the mechanism of organic A-V heart-block which will attempt to take into account the various phenomena which may be observed in this condition, with particular reference to the two cases described in this paper.

As is well known, the spread of the excitatory process or impulse through the heart is associated with a wave of negativity. The presence and movement of this wave is demonstrated both by electrograms obtained by direct leads from the heart of the experimental animal and by the electrocardiogram taken from the extremities. Conductivity is that property of the protoplasm which permits the spread of the impulse to occur. The experiments of Lillie[14] and others have led to the conclusion that the important factor in the conduction of the impulse is the excitability of the individual structural units which are traversed by the impulse. Each muscular element, as it becomes excited, undergoes a fall in its surface potential, and as a consequence an action current flows through it and the adjoining, positively charged and as yet inactive elements. The action current thus acts as the stimulus to these adjacent elements which in turn become negative and pass the impulse on to the elements beyond. It is apparent that if for any reason certain fibers reached by the action current are not sufficiently excitable, they will themselves fail to respond and will not transmit the impulse to other muscular elements. These fibers therefore constitute a block to the passage of the impulse. If such a group of fibers constitute merely an island in the auricular muscle, for example, the impulse will merely sweep around them, leaving them unaffected. If, how-

ever, these fibers are of sufficiently large mass and lie in the pathway between auricles and ventricles, A-V block will result.[15]

In considering the causes which may produce a block, it should be made clear that the decrease in excitability just mentioned is not the only factor which may result in failure of response. It is conceivable that the excitability of the fibers reached by an impulse may be normal, but that the action current may be too weak to stimulate. We suppose that as a matter of fact both alterations in excitability and in the intensity of the action current go hand in hand and are responsible for the phenomena of heart-block. It thus becomes imperative to examine more in detail the causes and nature both of the changes in excitability and of the effectiveness of the action current which acts as the stimulus.

In general it may be assumed that any condition which will lower the surface potential of the muscular elements will decrease their excitability. Such a decrease in potential may result from any injury whether due to traumatism, to infection, or to impairment of circulation resulting in diminished oxidations and an increase in the hydrogen-ion concentration. There are, of course, other possible causes of decreased conductivity (e.g., vagus influence upon the A-V node) but discussion of these would lead us too far afield, since our interpretation deals with block resulting from injury. In the same category with lack of oxygen is the influence of fatigue. Excessive activity, particularly of tissues already injured or anoxemic, will result in the accumulation of acid metabolites (e.g., lactic acid) with an increase in hydrogen-ion concentration and a consequent depression of excitability. That an increase in H-ion concentration depresses conductivity is shown by the work of Drury and Andrus.[16] Lewis and Master[17] in discussing fatigue phenomena in the normal dog's heart when a functional 2:1 A-V block is established at rapid auricular rates, give exhaustion of available energy-yielding food materials as a possible cause of the block. But whatever the cause of fatigue in normal tissue, we are inclined to regard the accumulation of acid metabolites as the more important cause of fatigue in injured tissue. Results obtained upon skeletal muscle when fatigued under conditions of lack of oxygen lend support to this view.[18] In this case complete fatigue supervenes long before the total supply of energy-yielding food material is exhausted.

Not only is excitability depressed by the conditions here mentioned, but there also occur the phasic variations in excitability with, and subsequent to, every response on the part of the tissues. Since, as already indicated, conductivity depends upon excitability, the former property of protoplasm undergoes the same variations as the latter. Trendelenburg,[19] Adrian,[20] and others studied the course of recovery of excitability following response in the muscle of the cold-blooded heart. When the response begins, the excitability drops to zero, where it remains

during the greater part of systole. This is the absolute refractory period. Toward or just at the end of systole, very strong stimuli may again excite the cardiac muscle, and as diastole progresses, the excitability returns, rapidly at first and then more and more slowly until, by the end of the phase of relaxation or slightly afterward the excitability is again at its resting level. This returning phase constitutes the relative refractory period. These results do not exclude the probability that with longer rest intervals there may be some slight further accession in excitability.[11] Junkmann[4] has recently reported that the duration of the absolute refractory period is greatly curtailed in fatigued cardiac muscle of the frog. Drury[12] states that in the dog's compressed auricular muscle the absolute refractory period is lengthened.

Results substantially the same as these for *excitability* have been reported for *conductivity* by Mines,[21] Ashman,[22] and Lewis and Master.[17] The clearest demonstration that the relative refractory period, as shown by the conductivity, is greatly prolonged in case of compression block, has been made by Ashman (Fig. 3-*A*). He found that not only may injury by compression greatly depress the resting conductivity in the turtle heart, but that the time required for recovery to a resting level, after an impulse has been transmitted through the compressed muscle, may be greatly increased over the normal. And it was also found that under conditions of injury and relative anoxemia, the conductivity may be further depressed by fatigue.

There remain to be discussed the effects which injury or previous activity may have upon the intensity of the impulse or action current. The latter, the result of the potential differences between active and inactive muscular elements, acts, as we have stated, as the stimulus to each successive element. Since, on this conception, the stimulus in case of conduction of the impulse is electrical in nature, it is necessary to state the conditions governing the effectiveness of such a stimulus. It is known that any stimulus, if it is to elicit a response, must (a) have a sufficient intensity, (b) last for a sufficient length of time, and (c) rise to a maximum intensity within a sufficiently brief time if it is a current of increasing intensity.[23] This last factor means that the *rate of change* in potential must be sufficiently great. Unless these conditions are fulfilled, the current will fail to cause a visible response. If *ABC* of Fig. 3-*B* represents a strip of cardiac muscle of which *B* is most injured, the potential on the surface of the elements of *B* will be lower, as indicated by the ± signs, than on the surfaces of *A* and *C*. Obviously, therefore, when *A* becomes negative in response to the arrival of the impulse from the sinus node, the potential difference set up between *A* and *B*, and therefore the action current, will not be so great as it would be if *B* were uninjured. For this reason, if the excitability of *B* is sufficiently depressed, *B* may fail to respond and acts as a block to the impulse. If it does respond, it will, because of the weakness of the

action current, respond less promptly than normally. Further, its fall in potential will be less than in normal muscle, the *change* in the existing potential will not be so great, and consequently *C* will respond less promptly. We believe that such factors are responsible for the delayed conduction seen in partial heart-block. As a result of the smaller potential differences, the rate of change in intensity will be less and this will tend still further to slow conduction. It will be unnecessary to discuss these factors further, but we must add that we appreciate the fact that we have only touched the surface of the subject in our presentation. For example, we have not discussed the action of the vagus nerve, which depresses conduction between auricles and ventricles, in spite of the probability, denied by Einthoven,[24] that it produces a positive variation in auricular muscle (Gaskell,[25] Meek and Eyster,[26] Garrey[27]) and the mechanism of block in this case may require a quite different interpretation.

One more point only need be mentioned before we come to our explanation of the mechanism of heart-block. That point is the phenomenon of conduction with a decrement. Drury[12] in a recent paper, found that an impulse entering a compressed region of the auricular muscle of the dog traveled more and more slowly as it penetrated farther and farther, and that it was likely to die out before reentering normal muscle beyond the compressed region. If it reentered normal muscle, it at once regained its normal velocity. This slowing up and presumable weakening of the impulse as it progresses in tissue, the conductivity of which is impaired, is called, after Lucas and Adrian,[28] conduction with a decrement. These authors postulated the phenomenon for an impulse traversing an anesthetized region of a nerve. More recently Davis, Forbes, Brunswick and Hopkins,[29] and also Kato,[30] denied the existence of conduction with a decrement for nerve, but it is difficult to find another reasonable interpretation for Drury's results. Certain applications of the conception of conduction with a decrement will be mentioned below.

AN INTERPRETATION OF THE MECHANISM OF HEART-BLOCK

Without committing ourselves to a definite statement as to the exact character of the injury responsible for atrioventricular heart-block, we may, for the purposes of discussion, regard the block as a region of damaged tissues between auricles and ventricles, diagrammatically represented in Fig. 3-*B* as *ABC*. *B* is regarded as a relatively narrow region of more intense injury, while the adjoining portions of *A* and *C* are less depressed in their conductivity. *B* is therefore a region in which (a) the resting excitability and conductivity are depressed, (b) the relative refractory period is much prolonged, (c) the intensity of the impulse is diminished and (d) there occur less rapidly those oxidative recovery processes which serve to prevent cumulative fatigue in

normal muscle at physiological rates of response. Similarly, the adjoining regions of *A* and *C* will be depressed, but to a lesser extent.

When the total degree of injury is relatively slight, the only abnormality in conduction observable will be a prolongation of the A-V interval. The reasons for this prolongation have already been given. As the degree of injury becomes greater an occasional auricular impulse will fail to reach the ventricle. For purposes of illustration we shall assume a 4:3 partial heart-block. The first auricular impulse after the dropped beat is, as is well known, transmitted relatively rapidly. In passing it leaves *B*, as well as the shaded portion of *C* in a refractory condition. The second impulse following the blocked impulse arrives at a time when *B* is relatively refractory and it is conducted more slowly. Since, moreover, the recovery processes in *B* are not so rapid as normal, there is cumulative fatigue, and the second impulse is transmitted more slowly for this reason also. The third impulse finds *B* still more fatigued, as well as again relatively refractory, and the A-V interval is consequently still longer. The fourth impulse arrives at *B* when its excitability is so depressed as a result of its relatively refractory and fatigued condition that *B* fails to respond and the impulse is blocked. Since *B* fails to respond, it has nearly double the usual time for recovery, both from its refractory state and from fatigue, so that the next impulse is transmitted most rapidly. It should be noted that, with a regular auricular rhythm, conditions in *A* will remain the same from cycle to cycle. *A* will thus share to some extent in producing the delay in conduction, but will play no part in causing the variations from cycle to cycle.

Lewis[31] (p. 177) states that in partial block of this type "the increase of the second interval (A-V) over the first [following the blocked impulse] is greater than the increase of the third over the second." This is understandable if it be assumed, as is highly probable, that the amount of catabolic chemical change occurring after the longer rest is greater than after the shorter rest. This would be a mass action effect. Thus the contribution to the cumulative fatigue of the first impulse after the pause would be greater than the contribution of the immediately following impulse. Another interpretation, which may mean the same thing fundamentally, is that the duration of the absolute refractory phase is longer after a long than after a short rest. Thus the absolute refractory period of *B* for the first transmitted impulse is longer than the subsequent absolute refractory periods. The recovery occurring during the relative refractory period will consequently not begin so promptly for the first as for the second impulse, and will therefore not have advanced so far when the second impulse reaches the block as when the third arrives. Junkmann[4] has shown that the absolute refractory period is shortened by fatigue, and Ashman* has

*Unpublished results.

found that during the onset of cumulative fatigue the electrogram at least, shortens progressively from cycle to cycle. This latter observation may indicate a similar shortening of the refractory period (Adrian[20]). If so, we may have a further reason for the phenomenon to which Lewis directs attention.

With still further increase in the degree of injury a 2:1 block may be established. With the mechanism above presented before us, little need be said in explanation of this degree of block. Here every alternate impulse arrives at a time when the excitability of *B* is such that it is unable to respond. As is generally recognized, 2:1 A-V block is

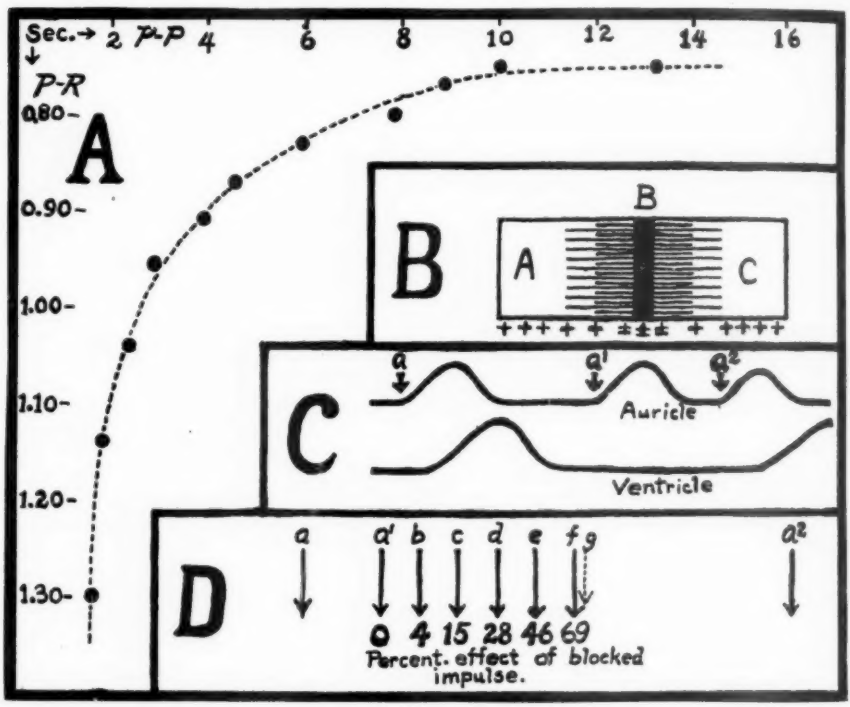

Fig. 3.—*A*. A curve showing recovery of conductivity derived from electrograms from the turtle heart compressed by clamp between auricles and ventricles. Ordinates, the P-R intervals in seconds; abscissae, the preceding P-P intervals in seconds. Note that with a short P-P, conduction time is long; with a long P-P conduction time is relatively short.

B. Diagram illustrating the injured and adjacent regions in A-V block. Discussion in text.

C. Myograms from the turtle heart. Discussion in text.

D. Diagram to illustrate the effect upon conduction of blocked impulses. The letters *a* and a^2 represent transmitted impulses. Blocked impulses interpolated between *a* and a^2, represented by a^1, *b*, *c*, *d*, *e*, and *f* have progressively greater effects upon subsequent conduction times. Impulse *g*, coming late after *a*, is transmitted. See text.

a relatively stable condition. This fact fits in well with our interpretation, since considerable change in the condition of *B* might occur without altering the 2:1 relation. Laurens[32] has observed in the turtle that when a sudden transition from a 1:1 rhythm (with prolonged

conduction time) to a 2:1 ratio occurs, there is a marked decrease in the A-V interval for those impulses which are transmitted, and the same phenomenon has been noted in clinical cases. This is exactly what we should expect, since with the onset of 2:1 block, the time for recovery of conductivity in *B* is doubled. That such a doubling of the rest interval will permit tremendous changes in conductivity to occur may be seen by reference to the recovery curves of conductivity in the compressed cardiac muscle of the turtle published by Ashman[22] (Fig. 3-*A*).

The fact just noted that a 1:1 rhythm may at times change directly to a 2:1 without the appearance of lower grades of block, such as 4:3, demands explanation. The cause seems to lie in the longer refractory period of region *B* for the first response after the longer rest. Thus every block impulse finds the recovery from the refractory period less complete than during the 1:1 rhythm. De Boer[10] has advanced such an explanation for analogous transitions, but attributed the effect to the longer ventricular refractoriness.

According to our view, the transition from 2:1 to 3:1 block, which would occur with still further injury, results when the duration of two auricular cycles is insufficient for the necessary recovery in *B*.

Thus far in our discussion, we have assumed a constant auricular rate. We must next consider the effects of a change in the auricular rate. It has been conclusively demonstrated that low grade partial block may be converted into block of higher grade or even into complete block as a result of a marked increase in the auricular rate. Erlanger[33] and also Lewis[31] have called attention to this result in cases of partial atrioventricular block experimentally produced in the dog. The converse phenomenon has been noted by Garrey,[8] who found that in the turtle heart a 2:1 rhythm, or even a higher grade of block, might be transformed to a 1:1 response as a result of vagus slowing. Our Case II illustrates this phenomenon particularly well (Fig. 4).

That in the usual clinical case of partial A-V block, exercise, amyl nitrite or other agent inducing augmented auricular rate does not usually lead to a greater degree of block is to be attributed to the fact that along with the loss of vagus tonus, which causes the acceleration of the auricle, there is a corresponding decrease in the vagus depression of conductivity in the injured conducting tissue. In discussing the mechanism of heart-block we are not primarily concerned with this secondary factor of vagus influence. We can state definitely that, barring nerve influences, or the supernormal phase,[34, 35, 36] an increase in auricular rate will always increase the degree of an existing organic A-V heart-block. This increase in degree may, of course, manifest itself merely as a prolongation in conduction time, but the effect is present. Let us suppose we have a patient with partial A-V heart-block (4:3 or 2:1) and that auricular fibrillation suddenly sets in. The block will become one of much higher degree, the ventricles are slowed,

and an idioventricular rhythm may even be established. To explain this result we look to the shaded area of *A* (Fig. 3-*B*). Each impulse as it arrives at *B* falls so early during the relative refractory period in the injured part of *A* that its intensity is markedly diminished and consequently *B* does not respond. It has been shown for nerve that an impulse set up during the early relative refractory period produces a weaker action current than the normal.[37] We are therefore justified in believing that all the impulses reaching *A* during the enhanced auricular rate will arrive at *B* with greatly diminished intensity. Perhaps if time were allowed for *B* to recover sufficiently, an occasional auricular impulse might be transmitted, but *B* is kept depressed by the slow idioventricular rhythm to which it will respond.

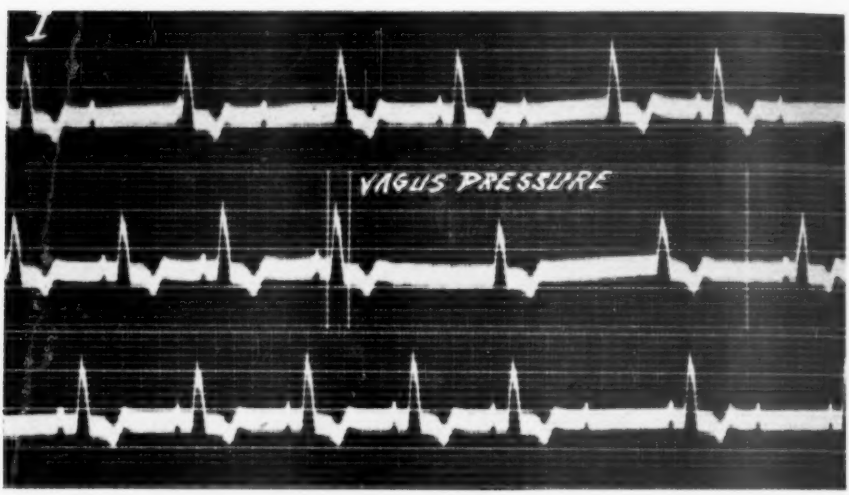

Fig. 4.—(Case II.) Lead I, continuous tracing. Pressure applied to left vagus at double signal line in strip two, and released at the single signal line. Note initial partial block with occasional idioventricular escape, suppression of the auricular pacemaker with vagus pressure and escape of the idioventricular pacemaker, the gradual acceleration in rate of the sinus node after release of pressure along with 1:1 rhythm, and the later appearance of 2:1 A-V block as the auricular rate increases further. Time in 0.2 and 0.04 seconds.

It should be noted that under these conditions *B* will not remain so narrow, but will widen to include most of the shaded area.

We have seen that the phenomena of low and high grade partial heart-block and of atrioventricular dissociation when auricular fibrillation sets in in a heart already exhibiting partial block, receive a ready explanation. We may mention certain other phenomena which likewise support our view of the mechanism of heart-block.

It is perhaps generally assumed that the blocked impulse in, for example, 4:3 A-V block is entirely without influence upon the subsequent A-V interval and that the reason why the A-V interval following the dropped beat is shorter is that the depressed conducting tissue has had a longer rest. To what extent this belief is true, as-

suming our interpretation of block to be correct, will be seen from our previous discussion. The A-V interval is shorter because regions *B* and *C* have rested longer; but *A* has had no rest. It is obvious therefore that if the blocked auricular impulse had been omitted, the following A-V interval would have been somewhat shorter, since *A* would have had a longer rest and the following impulse would have been transmited through *A* more rapidly. That the blocked impulse actually does leave a refractory state in the injured tissue and prolongs the conduction time for the subsequently conducted impulse has been shown most clearly by Ashman for the turtle heart.[22] The same thing was later demonstrated for the dog heart by Lewis and Master.[17] Fig. 3-*D* shows both the extent of this effect, as illustrated by averages of a considerable number of determinations, and the further fact that as the blocked impulse arrives at the block less and less prematurely after the previous transmitted impulse, its effect upon the next A-V interval becomes progressively greater. In the figure the arrow, *a*, marks the time of arrival at the compressed muscle of a transmitted impulse. If the next impulse to arrive is at a^2, the A-V interval will have a certain value which is taken as a measure of conductivity at that time. If an impulse had been interpolated between *a* and a^2 at the point shown by the arrow a^1, the interpolated impulse would have had no effect upon the A-V interval following. This is indicated by the 0 at the end of the arrow. If, instead of interpolating the impulse at a^1, the impulse had been sent in at *b*, the subsequent A-V interval would have been increased on the average about 4 per cent as much as by a transmitted impulse at *b*. Impulse *b* of course could not have been transmitted unless *a* had been omitted. Thus the number at the end of each arrow represents the average percentage effect upon conductivity of the blocked impulse arriving at the indicated interval after *a*, the effect of the transmitted impulse being taken at 100 per cent. An impulse at the dotted arrow, *g*, would have been transmitted.

Observations by Dr. W. E. Garrey on the heart of a dying rabbit (unpublished) seemed to show quite clearly that an impulse sweeping over the auricle was likely to be blocked at any one of several distinct points. The wave of contraction, at least, invariably stopped at one of these points and not at any point in the muscle. With the form of clamp used in the turtle experiments we are describing, there is every reason to believe that the degree of injury became progressively greater from the neighborhood of the auricular border of the block to the middle of *B*. We may therefore picture impulse a^1 (Fig. 3-D) as reaching an intramuscular block at or near the auricular border of *A* (Fig. 3-*B*) where it is blocked because of its prematurity. It thus leaves little or none of the compressed muscle refractory and does not appreciably influence the conduction time for impulse a^2. Impulses

b and *c* may be regarded, in consequence of the longer rest, as penetrating farther into region *A* and as therefore producing a slight effect on the conduction time for a^2. In a similar manner impulses *d*, *e* and *f* penetrate still further to other intramuscular lines or points of block, leave a more extensive region in a refractory state, and consequently their effect is greater. It will be evident from this description that the breadth of region *B* is considered as varying with the cardiac rate. A difficulty is encountered in accounting for the great effect of impulse *f*, an effect averaging 69 per cent of that of a transmitted impulse, when, on theory, its effect should be rather less than 50 per cent. If, however, *f* succeeds in traversing most of region *B* the result is understandable. It is to be noted that this interpretation does not involve conduction with a decrement.

Lewis and Master[17] have proposed an interpretation of the greater effect of the later blocked impulse which depends upon conduction with a decrement. If such conduction be a fact, then the impulse *f* might pass regions *A*, *B* and part of *C* before being extinguished, and thus its pronounced influence upon conductivity is to be expected. Were it not for one further observation made by Ashman, we should be inclined to accept this interpretation. The observation in question is illustrated in Fig. 3-*C*. Here *a* represents the last transmitted impulse; a^1 the blocked impulse, which may arrive so late as just to fail of transmission to the ventricle; and a^2 the next transmitted impulse. It will be noted that the interval a-a^1 is considerably greater than the interval a^1-a^2. And yet a^2 is transmitted while a^1 is blocked. This result is difficult to explain on the basis of conduction with a decrement, since a^2, coming early after a^1, might be expected to have experienced a greater decrement than a^1. That it did not is proved by its transmission. On the basis of the mechanism of block herein presented we must assume that impulse a^2 not only penetrates to the same intramuscular block reached by a^1, but, finding the region beyond (*B*) more fully recovered, causes it to respond and thus makes its way to the ventricle. The failure of a^1 to reach the ventricle, and the success of a^2 may perhaps be interpreted as due in part to the longer absolute refractory period for *a* which follows a longer rest interval and a shorter one for a^1.

We do not by any means wish to go on record as denying the existence of conduction with a decrement. But we have experienced more difficulty in interpreting the various phenomena of heart-block on that assumption than without it. We wish to state further that we have not failed, in developing our conception of the mechanism of heart-block, to consider phenomena other than those which can be mentioned in a paper of this scope.

A CONSIDERATION OF THE PHYSIOLOGICAL SIGNIFICANCE OF THE ELECTROCARDIOGRAMS

One of the most characteristic phenomena in experimental partial atrioventricular heart-block is the effect, previously discussed, of increasing the auricular rate. In human cases the effect is usually complicated by the fact that an increase in the auricular rate due to release of the sinus node from vagus influence is associated with a corresponding release of the tissues in which conductivity is impaired. The two cases herein reported afford a sharp contrast when the effects of increased auricular rate are considered. In Case I there appeared to be little or no vagotonic depression of conductivity in the injured tissue, while in Case II this effect is fairly well marked. As a consequence in Case I when a 1:1 rhythm is present a sufficient increase in the auricular rate invariably produced an increase in the degree of block, so that the idioventricular pacemaker took over the function of determining the ventricular rhythm. In Case II on the other hand an acceleration of the auricles, within limits, resulted in an acceleration of the ventricles, the idioventricular rhythm previously largely in control of ventricular rate being suppressed. A still further increase in auricular rate, however, resulted in a 3:1 block with a decrease in ventricular rate at the point of transition from 2:1 to 3:1, but the latter rate was still too high to allow the idioventricular pacemaker to become active. The appearance in this case of 3:1 block shows that the decreased vagus influence on the conducting tissues associated with the acceleration of the sinus node, could not compensate fully for the tendency to an increased degree of block with high auricular rates. Yet even in this case, vagal compression did not so greatly depress conductivity as to prevent the appearance of a 1:1 rhythm coincident with the marked auricular slowing. In the turtle a similar effect of vagus slowing has been noted by Gaskell[25] and by Garrey.[8] The clearest evidence in Case II that the vagus contributed to the depression of conductivity in the junctional tissues was a decrease at times in the P-R interval when the auricle was more rapid.

Another striking difference between our two cases is in the length of the P-R intervals. In Case II the P-R intervals varied from 0.18 to about 0.25 sec. At their shortest, therefore, they were almost within physiological time limits. In our Case I, in contrast with Case II, we have very conclusive evidence of conduction times up to 0.75 sec., the average on the same date being 0.70 (Figs. 5 and 6). But electrocardiograms from the same patient taken recently show conduction times extending to a full second and over (Figs. 7 and 8). The evidence for conduction on these electrocardiograms seems conclusive to us, as we shall show in a later paper. If our interpretation be correct, these P-R intervals far exceed any previously reported from electrocardiographic evidence. The longest previously

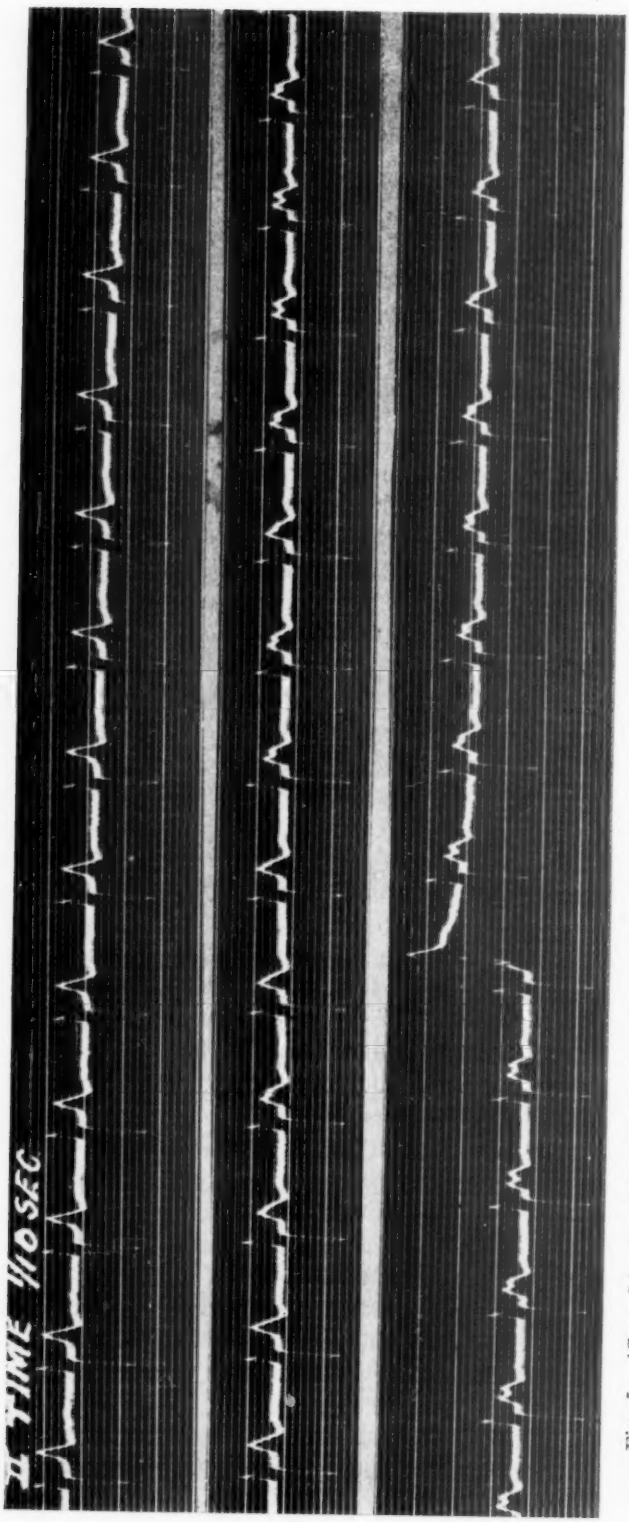

Fig. 5.—(Case I.) Lead II, continuous tracing. A period of 1:1 rhythm associated with a slow auricular rate (62 to 67 per minute). The conduction time varies from an average of 0.69 second at the slower rate (62) on the first strip, to an average of 0.72 second at the faster rate (67) at the beginning of the third strip. Note the resultant shift in the position of the P- and T-waves. Time in 0.1 and 0.02 seconds.

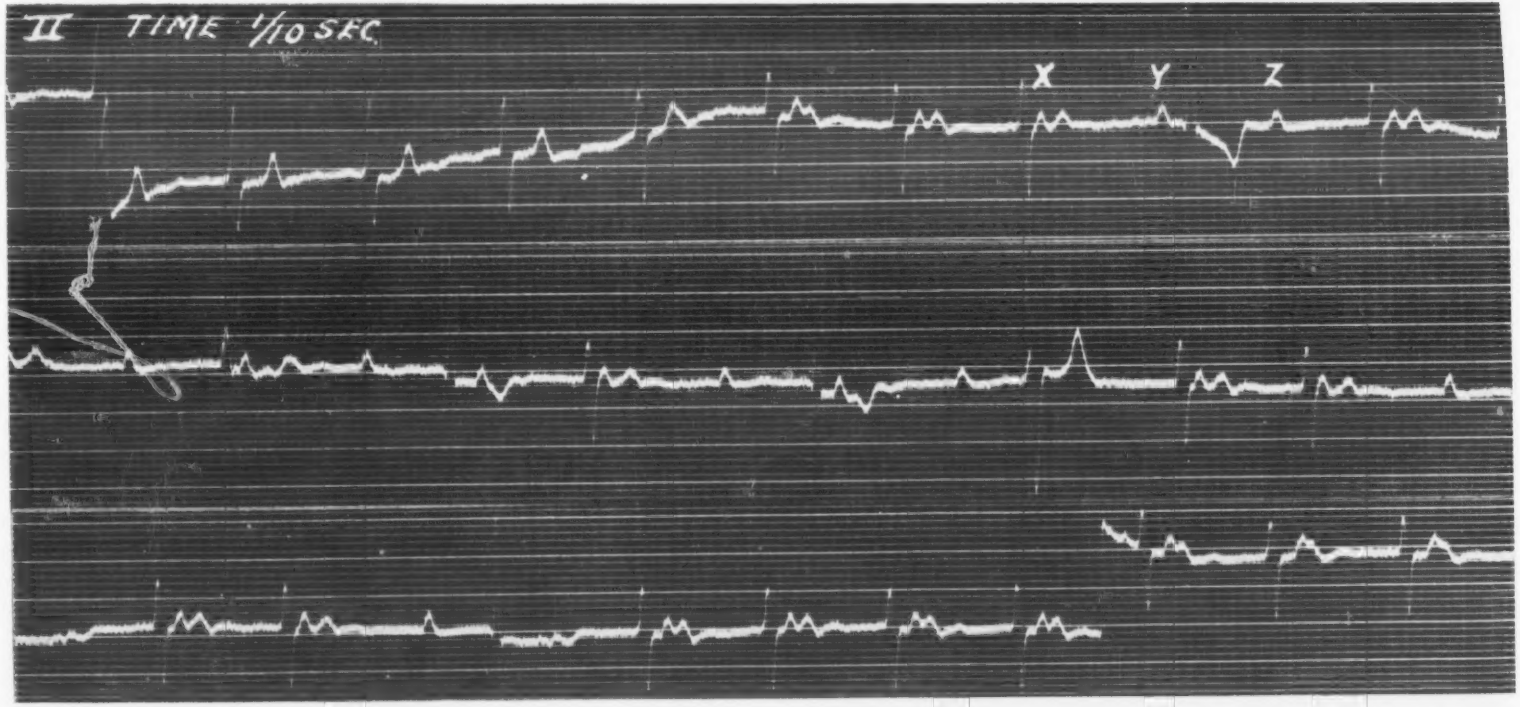

Fig. 6.—(Case I.) Lead II, continuous tracing. Strip one shows a 1:1 rhythm with acceleration of the auricles until at *X* the P fails to be transmitted. Before P-wave *Y* can be transmitted, the idioventricular pacemaker escapes, while P-wave *Z* is transmitted in 0.68 second, the shorter conduction time being associated with the longer rest in the ventricular side of the block (cf. Fig. 5). Strip two, and the first third of strip three show ventricular complexes of idioventricular (two foci) and of supraventricular origin. In the rest of strip three, the 1:1 rhythm is resumed as a result of a slowing of the auricular rate from about 70 to about 66 per minute. Time, 0.1 and 0.02 seconds.

reported in the literature were in a case of Thayer's.[38] In his electrocardiograms (Plate IV) the P-R intervals varied from 0.64 to 0.72 sec. with the average at 0.68. One interval reaches 0.88, but the evidence for conduction, while good, is not quite conclusive for this complex. The P-R of the preceding complex, 0.23 sec., interpreted as conduction time by Thayer, almost certainly does not represent true conduction as is evidenced both by its relatively extreme brevity and by the form of the ventricular complex (Y). As has been sufficiently shown above, the abnormal P-wave (W) would not have been without influence upon conductivity, which adds to the improbability that the 0.23 sec. interval is a true conduction interval. The R-R interval preceding Y, as well as the following R-R interval, is the longest in the figure. This fact likewise argues strongly that Y is idioventricular, and makes it possible that the following complex is also idioventricular, although originating from a different focus. If so, then the P-R interval of 0.88 does not represent a transmission interval. We agree with Thayer that the other auricular impulses are transmitted.

Several explanations[31] have been advanced for variation in conduction time in different cases as is illustrated by the difference between our Cases I and II. Conduction is normally slow through the A-V node and most rapid in the Purkinje fibers. Thus the locus of the injury may well play a part in determining that in one case A-V block may appear even without conspicuous premonitory lengthening of the P-R intervals, while in other cases marked prolongation of the P-R interval may appear before there is any failure of ventricular response. We propose the explanation that, other conditions being equal, a prolonged conduction time is a result of the existence of a greater breadth of injured tissue through which the impulse must make its way; a less prolonged conduction time, a shorter stretch of injury. This explanation is, we believe, most consistent with the mechanism of heart-block which we have proposed above. Drury's[12] experiments lend considerable support to this view. Those cases of complete block of sudden onset, not ushered in by premonitory lengthening of the P-R interval such as one reported by Wilson and Herrmann,[39] we are inclined to believe may depend upon a different phenomenon, that of the supernormal phase in conductivity, a phenomenon which will be discussed in a forthcoming paper.

We have said that there can be no doubt that the P-R intervals of 0.70 sec. and over in Case I were examples of actual conduction, although the reader may question whether the 1:1 rhythm shown in Figs. 5 and 6, is actually such, or whether we are not here dealing with an idioventricular rhythm of unusual rapidity, and that the auricular systoles may be forced to keep pace with the ventricles as a result of some influence of the latter upon the auricular pacemaker. This case did in fact show such influence to a marked degree at other times. The

authors are convinced, however, that there can be no doubt of the correctness of their interpretation in this instance. There are at least four reasons for believing that the auricular impulses are transmitted. (1) When the heart rate slightly accelerates (during the 1:1 rhythm) the P-waves fall closer to the QRS. This is due directly to the more

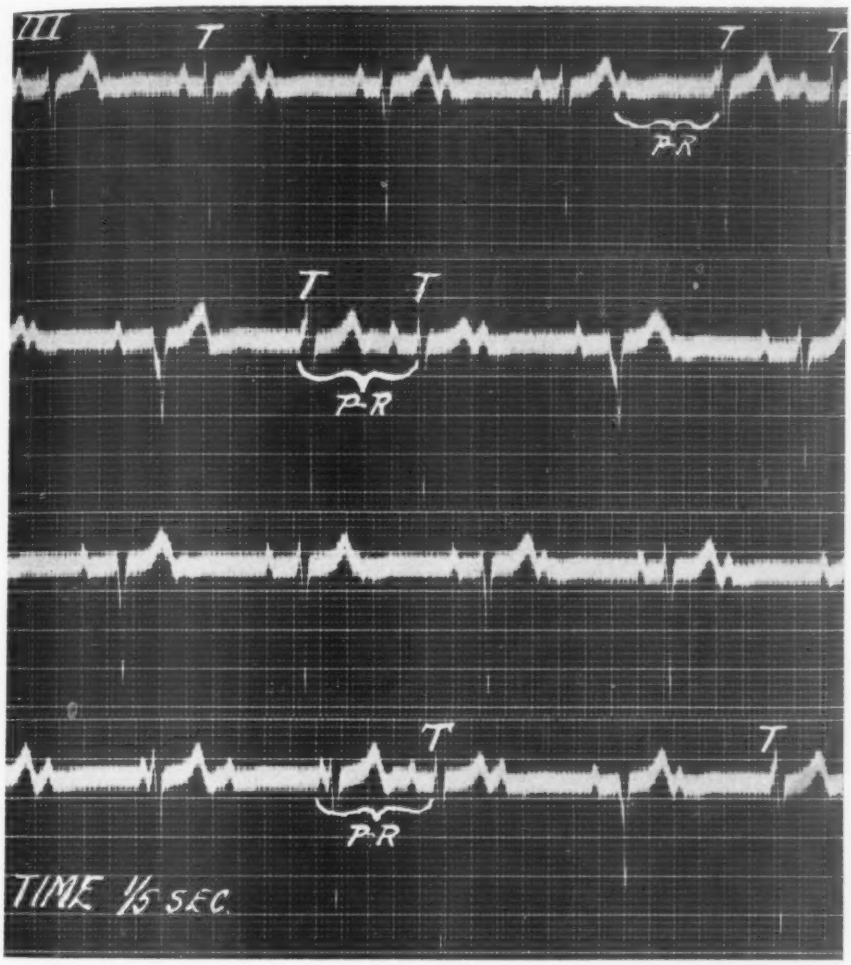

Fig. 7.—(Case I.) Lead III, continuous tracing. Complexes 2, 5, and 6 of strip one; 2 and 3 of strip two; and 3 and 5 of strip four are responses to auricular impulses, while certain others, such as 1 and 4 of strip two are definitely idioventricular. The remaining complexes are combinations or not definitely established. The P-R intervals range from 0.76 to 0.88 second. Since the P-R intervals exceed the P-P intervals in length, a second P-wave falls between each transmitted P and the consequent ventricular complex. (Cf. Figs. 5 and 6 taken about 30 days earlier.) The peculiarities in conduction here shown we attribute to a supernormal phase. Time in 0.2 and 0.04 seconds.

rapid rate and to a slight prolongation of the P-R intervals. If the discharge of the sinus impulse were due to a mechanical tug, for example, the P-waves should fall later, not earlier, during ventricular

systole, since with the more rapid rate less time would be permitted for the recovery of excitability in the sinus node. (2) The P-R interval is definitely increased with increased rate. This is, of course, the anticipated result if the impulses are actually transmitted. (3) The rate of the ventricle likewise militates against the argument for a ventricular influence upon the pacemaker since the ventricle is much more rapid than it was in the same patient during periods of complete heart-block. (4) The transition from the 1:1 ratio to a higher degree of block argues very strongly for transmission, since the transition is always associated with an increase in auricular rate. A fifth reason

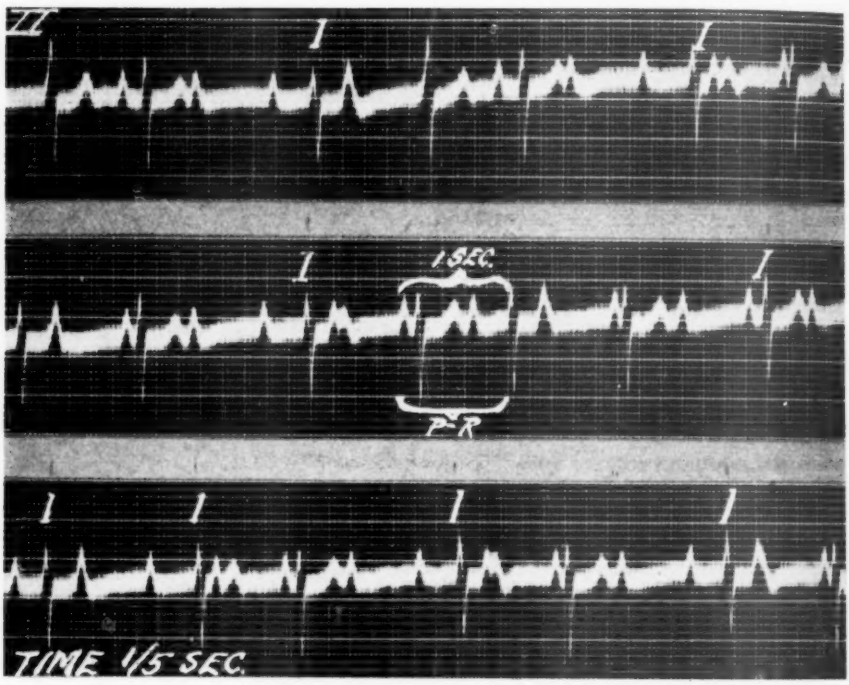

Fig. 8.—(Case I.) Lead II, continuous tracing, taken on the same day as Fig. 7. See legend for the previous figure. The idioventricular complexes are marked I. All the rest are believed to be responses to auricular impulses. The P-R intervals vary from about 0.72 to 1.00 second. The conduction time of 1.00 second is marked with a bracket (strip two). An analysis of the whole of our curves of this date supports the view that the impulses are transmitted, the peculiarities being due to a super-normal phase. Time in 0.2 and 0.04 seconds.

for transmission may perhaps be found in the fact that the form of the ventricular complex changes when the abrupt slowing in ventricular rate takes place, but we regard this reason as not necessarily valid.

The argument is possibly somewhat more doubtful for those P-R intervals, recorded on a later date, which range upward to a full second.* (Figs. 7 and 8.) Here the ventricular responses come in groups of two, three or four, separated by pauses. We are inclined to believe that

*Later studies have convinced us that even the extremely long intervals represent conduction times.

the first of these complexes in each group is usually idioventricular, although originating above the bifurcation of the A-V bundle, while the other systoles of the group are initiated by auricular impulses. We believe further that the peculiarities in conduction displayed by this patient, other than the extreme length of the P-R intervals, are due to a supernormal phase in conductivity, a phenomenon already reported for clinical cases by Lewis and Master[36] and by Wolferth.[40] The patient had not manifested any clear evidence of this phenomenon on earlier dates. We intend to publish our analysis of the curves demonstrating the supernormal phase as a later communication. The P-R intervals on this date varied from 0.70 to 1.01 sec. In one doubtful instance the P-R may have fallen as low as 0.54. The average was 0.83 or 0.84, although P-R intervals as high as 0.90 and above were very frequent.

The effect of auricular rate upon the degree of block, and the greatly prolonged P-R intervals have been discussed in detail; the former because of its fundamental importance and because it is a factor not ordinarily considered in clinical papers; the latter because of the unprecedented length of the intervals. There remain a large number of phenomena manifested at one time or another by these hearts which we shall discuss as they are related (1) to impulse formation, (2) to conduction, and (3) to combination complexes resulting from the simultaneous spread of idioventricular and supraventricular impulses through the ventricle.

We have already alluded to the influence, clearly seen in our two cases, which the ventricle exerts in causing a premature discharge of impulses from the sinus node. This influence is discussed by Lewis,[31] (p. 178) and by Wilson and Robinson.[41] The shortening of the auricular cycle during which the ventricular systole falls, or regarded conversely, the lengthening of the cycle during which no ventricular systole falls in cases of 2:1 or of complete block, is in places very conspicuous in Fig. 7.[42] The view of Erlanger and Blackman[43] that the variation in the duration of the auricular cycles is due to reflex vagus influence is opposed by the fact that when two ventricular systoles occur in rapid sequence both auricular cycles may be shortened (Fig. 7, lower line). This observation at the same time strengthens the view of Wilson and Robinson that the premature discharge is the result of a mechanical tug or other direct influence from the ventricle.

A remarkable shift in the location of the pacemaker from the sinus to the A-V node was observed in Case II. Fig. 9 shows a gradual acceleration of the sinus rhythm, due to release from vagus pressure, from cycle lengths of about 1.2 sec. to nearly 1.0 sec., until finally a single impulse fails to be transmitted; and then a pause ensues which is strongly suggestive of a sinoauricular block. After 1.4 seconds some ectopic focus, presumably the extreme upper end of the A-V node,

escapes and becomes the pacemaker. The surprising feature of this change of pacemaker is that the less rhythmic A-V node usurps the rôle of pacemaker from the more rapid sinus node. If we assume that the nonappearance of the P-wave at the expected instant before the A-V rhythm begins is due to sinoauricular block, we can attempt an

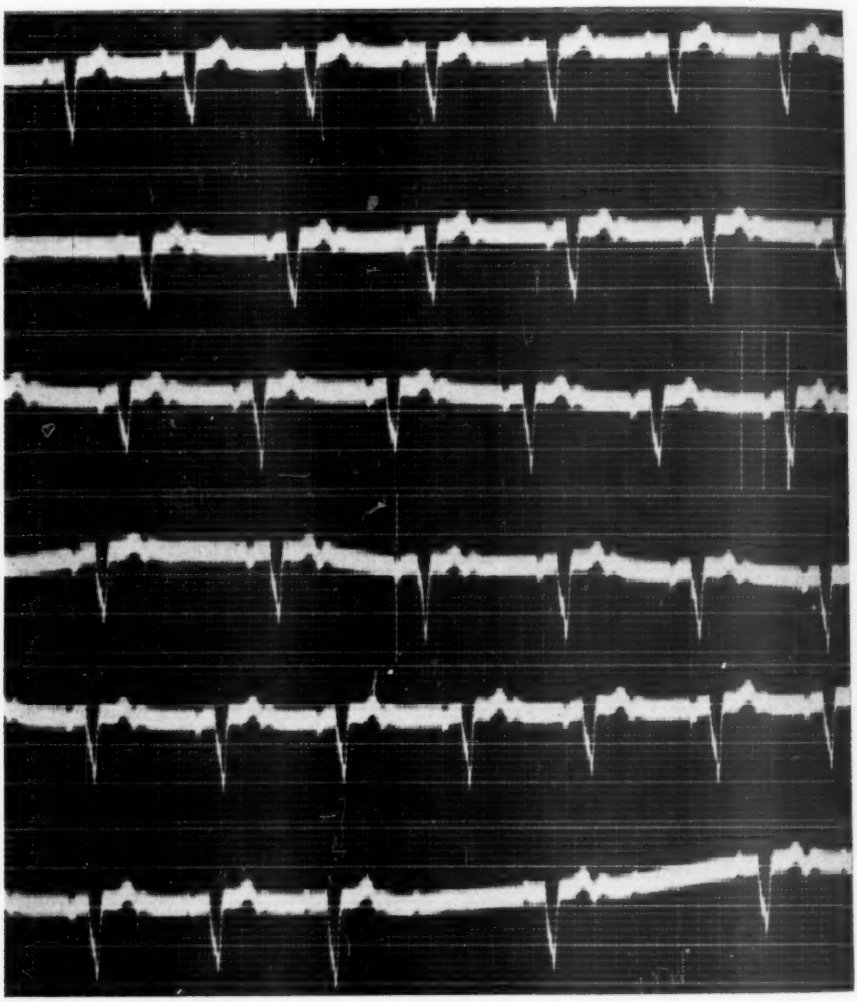

Fig. 9.—(Case II.) Lead III, continuous tracing, showing a shift in position of the auricular pacemaker associated with acceleration of the sinus node. Note the change in the P-waves. Right vagus pressure applied at the three signal lines at the end of strip three and released at the single signal in strip four did not cause a reshift in the pacemaker. Further description and discussion in the text. Time in 0.2 and 0.04 seconds.

explanation of the maintenance of the slower A-V rhythm in the face of the probable existence of a more rapid sinus rhythm. In view of the direct analogy between this phenomenon and the parasystolic theory proposed by Kaufmann and Rothberger[44] to account for extrasystoles,

it deserves to be emphasized. In Fig. 11, the assumed sinus impulses are designated by the row of downwardly directed arrows. The underlying horizontal line represents the region of the postulated sinoauricular block, and the upwardly directed arrows the A-V impulses. So long as the block, which may be largely the result of vagus influence, persists, it will be seen from the time relations of the arrows that no sinus impulse is likely to break through into the auricle because whenever the sinus impulse arrives at the block it finds as great or nearly

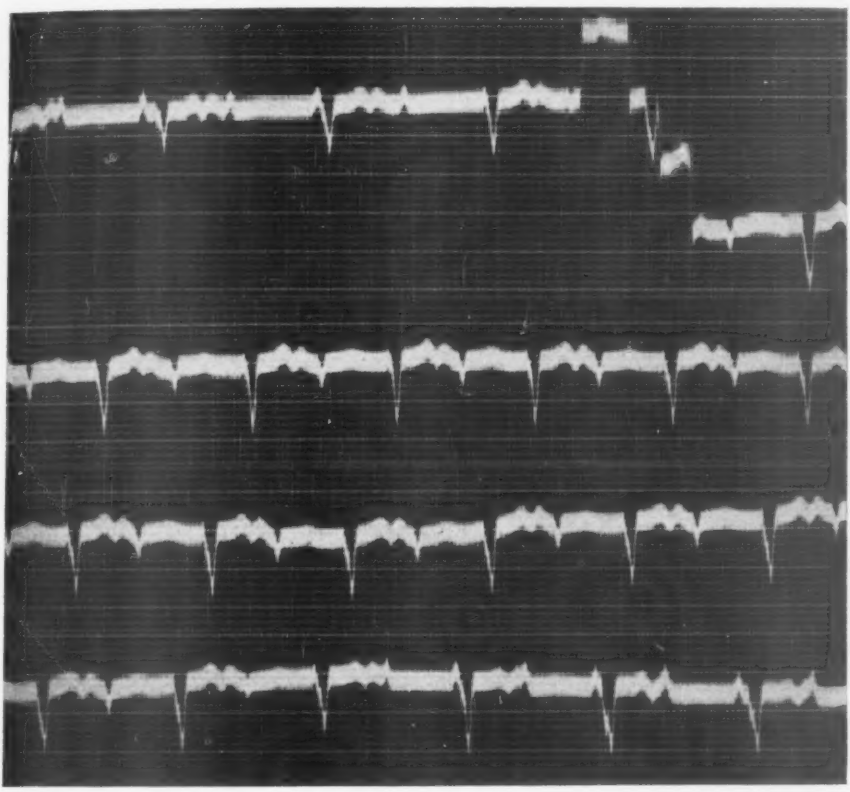

Fig. 10.—(Case II.) Lead III, continuous tracing, showing a shift in position of the auricular pacemaker associated with a slowing of the sinus node. Described in detail in the text. Time in 0.2 and 0.04 seconds.

as great, a refractory state at that region as at the time of blocking of the sinus impulse immediately preceding the beginning of A-V rhythm. This explanation is facilitated if it be assumed that the A-V impulse, *p*, broke through the block and disturbed the sinus rhythm. That this might happen is the more probable since the refractory state on the auricular side is less marked than on the auricular side because of the slowness of the A-V rhythm. If *p* breaks through the conditions will be restored to those existing at *b* and the sequence of events will be repeated. The transition back to normal rhythm comes at a time

when the A-V rhythm is accelerating and the heart, including the region of sinoauricular block, is escaping from vagus influence. It is of interest to note that the A-V rhythm was maintained throughout the vicissitudes of pressure on the right vagus. The pressure was applied at the time of the three vertical signal lines at the end of the third strip of electrocardiogram, and was released at the single signal on the next strip. Several other similar shifts of the pacemaker were recorded on this same date.

Fig. 10 illustrates an entirely different kind of shift in the location of the pacemaker. It was obtained from the same patient and on the same date as Fig. 9. The first line shows an acceleration of the sinus rhythm until the auricular cycle lengths become 0.8 second and a 2:1 block is present. Just before the standardization (upper line), an auricular combination complex appears. This is caused by the escape of a focus presumably near the middle of the A-V node, and the retrograde impulse meets the sinus impulse in the auricular muscle. The next S-wave contains, we believe, an inverted P which is lost. Midway between this S and the last S on the line is an inverted P. We interpret this curve as an example of the escape of an atrioventricular rhythm,

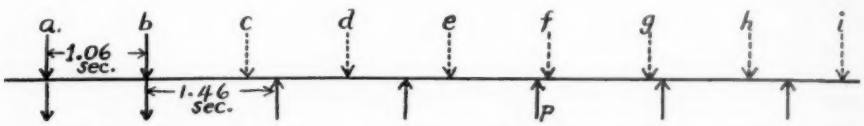

Fig. 11.—Schema to represent the changes in location of the pacemaker shown in Fig. 9. The letters *a* and *b* represent transmitted sinus impulses; *c, d, e, f, g, h,* and *i,* supposed blocked impulses. Upwardly directed arrows, impulses from the ectopic focus. Horizontal line, the postulated sinoauricular block. Discussion in text.

and believe that every other impulse fails to reach the ventricles while the alternate impulses are lost in the ventricular complexes. In this example of A-V rhythm, unlike the former, the A-V node takes over the function of the pacemaker because, temporarily, its rhythmicity exceeds that of the sinus node.

Changes in the position of the idioventricular pacemaker also have been observed during periods of complete or nearly complete block, but nothing of especial interest has come to light regarding them. Another interesting occurrence was the appearance of ventricular extrasystoles, usually from a single focus which was not identical with the idioventricular focus responsible for the slow rhythm during complete block (Figs. 12 and 13). Fig. 13 shows an unusual form of ventricular extrasystole. During the progress of an idioventricular rhythm, apparently originating high up in the bundle or in the A-V node and interrupted at times by responses to transmitted impulses (upper line, complexes two and six), there appears an extrasystole of almost normal complex form. It contrasts strongly with the other ventricular complexes which demonstrate the presence of a right bundle branch block. Barring a marked

supernormal phase for conduction through the right bundle branch, a condition for which we have no evidence in this case (II), the only possible explanation of the form of this complex seems to be that it arises in or near the injured muscle of the right bundle and arrives simultaneously, via normal pathways, at the musculature of the right and left ventricles. The alternative suggestion, that two idioventricular

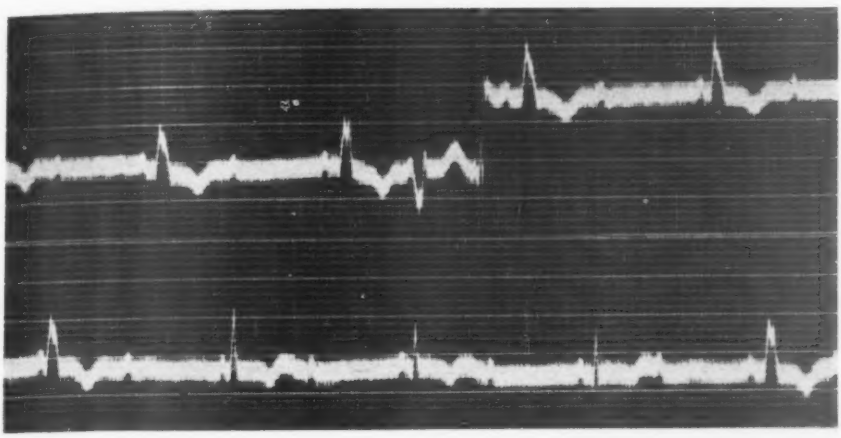

Fig. 12.—(Case II.) Lead I, continuous tracing. A single right ventricular ectopic beat and a 2:1 A-V block are shown in strip one. Strip two shows combination complexes with unusually narrow QRS groups. Discussed in text.

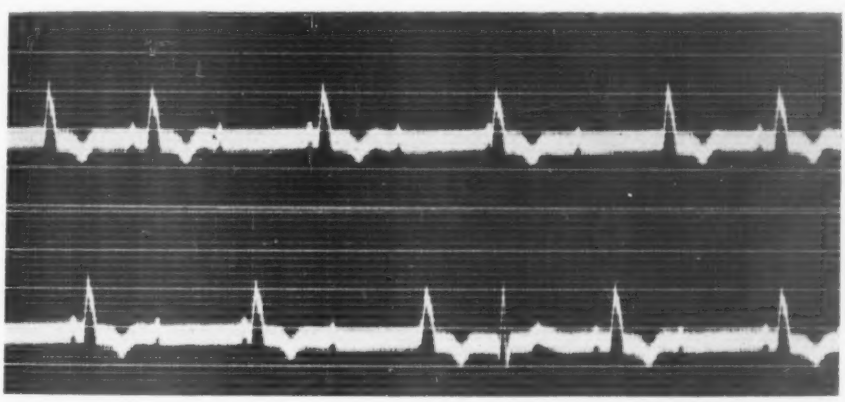

Fig. 13.—(Case II.) Lead I, continuous tracing, showing right bundle-branch block and idioventricular rhythm interrupted at frequent intervals by responses to impulses of supraventricular origin. In strip one, complexes 2 and 6; in strip two, complexes 5 and 6 are transmitted. The ectopic complex 4 in the lower strip is discussed in the text.

foci, one in each ventricle, may have discharged simultaneously, is, in view of the very infrequent occurrence of ectopics in this patient at this time, in the highest degree improbable.

Vagus pressure in Case II, in addition to producing the conditions favorable to the escape of the atrioventricular node which have been

discussed, usually caused at first a complete suppression of the pacemakers, both sinus and atrioventricular, and thus permitted the escape of the idioventricular focus. As the sinus rhythm returned following vagus pressure, it was at first slow and a 1:1 ratio between auricles and ventricles was nearly always established. This in turn gave way to a 2:1 A-V block as the auricular rhythm became still more rapid, although, since the rhythm was still somewhat slowed, there were later frequent escapes of the idioventricular pacemaker. That these escapes did not appear at once with the return to 2:1 rhythm we attribute to fatigue of the idioventricular pacemaker by the preceding relatively rapid ventricular rate. These effects of vagus pressure are illustrated in Fig. 4. With a further acceleration of the sinus rhythm a simple 2:1 block was always exhibited by this patient, and on one occasion, after exercise, the auricular rate went to about 120 per minute and a 3:1 block appeared.

As a result of the changes in rate of the auricles which occurred in both patients, but especially in Case II, it frequently happened that there was escape of the idioventricular pacemaker at the same instant that the ventricular muscle was being invaded by the impulse of supraventricular origin. The resultant ventricular complexes were consequently modified in form (Figs. 12 and 14). Such combination complexes have been produced experimentally in the dog by Wilson and Herrmann.[45] These investigators first instituted a bundle-branch block and obtained electrocardiograms demonstrating the forms of the ventricular complexes resulting from stimulation of the myocardium of the ventricle on the side of the block at various times during the spread of the physiological impulse through the opposite ventricle. Since in our patient the idioventricular impulse arose in different instances at different times during the spread of the supraventricular impulse, combinations were produced of the various types figured by Wilson and Herrmann. Recently Korns[46] has published electrocardiograms of a clinical case which show combination complexes which he interprets as resulting from the spread in different time relations of supraventricular impulses through the two bundle branches.

A comparison of the early and late electrocardiograms from Case I shows that there was a gradual shortening of the QRS group of the idioventricular complexes from about 0.14 second to 0.10 or even less (Figs. 1 and 8). Two possible explanations of this change suggest themselves. The change may be due to a gradual shift in the location of the idioventricular pacemaker from a position in the upper part of the left bundle branch to a higher location. In favor of this interpretation is the simultaneous gradual acceleration of the idioventricular rhythm suggesting a shift in the pacemaker from a region of lower to one of higher rhythmicity. The other explanation is that the change from the

earlier, broader complexes to the later form may have resulted from a gradual recovery of the specialized tissues of the ventricle from a condition which depressed their conductivity at the same time that it depressed the rate of the idioventricular pacemaker. In favor of this view is the fact that the auricular pacemaker showed an acceleration in rate along with the idioventricular.

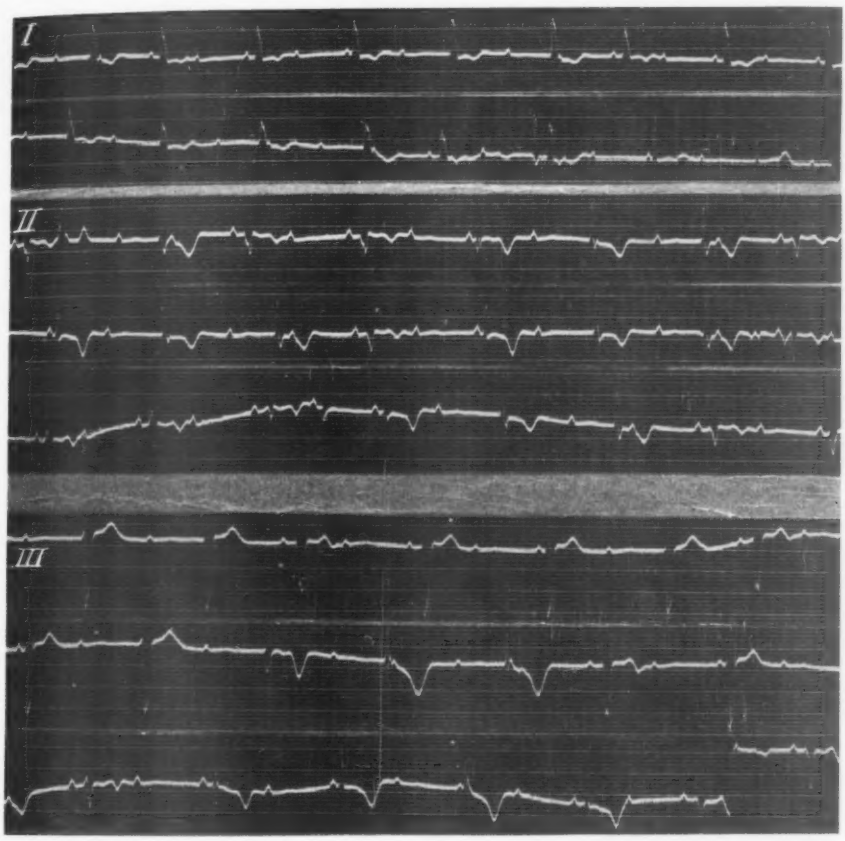

Fig. 14.—(Case II.) Lead I, continuous tracing, strips one and two. Lead II, continuous, strips three, four and five. Lead III, continuous, strips six, seven and eight. Right bundle-branch block and 2:1 A-V block with frequent right ventricular escapes resulting in combination complexes of all types. Time, 0.2 and 0.04 seconds.

In conclusion we may call attention to the existence of apparently complete retrograde block in both cases. Retrograde conduction (i.e., V-A conduction) was nowhere seen in our long series of electrocardiograms in spite of the frequent occurrence of idioventricular beats which might frequently have been transmitted to the auricles had retrograde block not been present. This observation is consistent with the fact which is gaining recognition that complete V-A block will appear earlier than A-V block when the conducting tissues are injured.

SUMMARY

1. Two clinical cases of heart-block are presented which show unusually significant therapeutic responses. The spectacular results of barium chloride therapy as well as the effects of other drugs are discussed in detail.

2. An explanation of the mechanism of heart-block is offered.

3. Analysis of the electrocardiograms brings to light many interesting abnormalities in the cardiac mechanism in the interpretation of which facts of fundamental physiological importance are both applied and illustrated.

The effects upon conduction of changes in the auricular rate; the occurrence of P-R intervals of unprecedented length, ranging up to a full second; remarkable shifts in the location of the auricular and also of the ventricular pacemaker, and ventricular combination complexes of various types are illustrated and discussed.

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GROSS AND MICROSCOPICAL ANATOMY OF THE BLOOD VESSELS IN THE VALVES OF THE HUMAN HEART*

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THE embolic theory of valvular endocarditis as first announced by Koester[1] in 1878 has been attacked by a number of investigators because it has never been conclusively proved that the blood vessels which are occasionally found in the valves of the adult human heart may not be of postinflammatory origin.

Luschka[2] (1852-1863) described the presence of vessels in the auriculoventricular and semilunar valves, but failed, as did the later writers, to show that these valves were normal, or if pathological, that the vessels preceded endocarditis. He claimed that all cardiac valves were vascularized normally, and that vessels passed through the chordae tendineae to anastomose with those of the valves.

Coën[3] (1886) disagreed with Luschka, denying that the chordae tendineae and semilunar valves normally possessed blood vessels.

Langer[4] (1880 and 1887) observed that fetal valves contain musculature and blood vessels, but that these regressed before birth. He was of the opinion that when blood vessels were found in adult heart valves they were of postinflammatory origin.

In 1887 Darier[5] formulated the difficulties in accepting the existence of vessels in the valves of adult hearts in terms of a series of critical questions which led him to the conclusion that all vascularized valves in adult hearts owe their vessels to the existence of inflammation, with the production of granulation tissue vasculature. He was largely led to this conclusion by the observation that all valves other than those of fetuses which he found vascularized were invariably the seat of an inflammation. Koeniger[6] (1903) and Odinzow[7] (1904) came to similar conclusions.

Rappe[8] (1904) was the first to adduce evidence that the vessels seen in valves other than those of the fetus were preinflammatory. His views were based largely on the fact that the vasculature of the valves showed a remarkably constant structure, that if these vessels were of inflammatory origin one ought to expect a large number of them to descend in the valve leaflets more or less parallel to one another in order to vascularize the inflamed area, and that it was difficult to believe that granulation tissue blood vessels should spring so far from

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the source of inflammation. He was, however, puzzled by the fact that the presence of blood vessels seemed to be in all his cases associated with an endocarditis and came to the curious conclusion that "this typical course in pathological conditions just as in normal valves dims our vision and yet we have no explanation."

In 1917 Bayne-Jones[9] injected a series of fourteen human hearts and obtained injections in the valves of three. He concluded that blood vessels normally occur in valves and that his failure to inject them in all the hearts was probably due to imperfect technic and lack of proper conditions. In this work Bayne-Jones selected what appeared grossly to be normal hearts.

In 1921 one of us (Gross)[10] published the results of his observations on one hundred human hearts. These hearts were injected by a standardized technic which enabled him to submit each specimen to exactly the same technical procedure. The observations on the vessels were made by means of stereoscopic x-ray plates, dissections and rendering the valves transparent, and by microscopic studies. The conclusions arrived at were in part as follows:

1. All fetal valves possess musculature and vasculature.
2. Usually sometime before birth the musculature and vasculature undergo regression.
3. In a small percentage of individuals there is a persistence of blood vessels to adult life and this persistence renders the individual predisposed to embolic valvular endocarditis.
4. The aortic leaflet of the mitral valve is the last to lose its vasculature and is the most frequent seat of endocarditis.
5. Those valves which did not show vessels grossly (when injected by this method) did not show vessels microscopically.

No mention is made, by the last two workers cited, of histological examinations of musculature and valves in the hearts with successful valvular injections, to prove that any of the injected valve leaflets were normal or that the blood vessels found were of preinflammatory origin.

The present status of the problem is therefore that no satisfactory proof exists that valves other than those of the fetus can possess vessels which do not owe their origin to granulation tissue, although this view is strongly suggested by the deductions made by Rappe and by Gross.

The present work was undertaken in an attempt to gain further information upon this question, and this report deals with observations which were made on eighty-five human hearts representing specimens from fetuses, newborn and all age periods up to and including the ninth decade.

The method which we pursued was to inject the hearts under standardized conditions with a technic already described by one of us.[10] In brief, this consisted of injections of the coronary arteries, and in some cases the coronary sinus, with barium sulphate gelatine, clearing

the hearts to render them transparent, dissections and microscopic sections. The latter were taken from each valve of the heart, from numerous parts of auricular and ventricular musculature, the tips of the papillary muscles, the root of the aorta, the endocardium and pericardium. The sections were stained with hematoxylin and eosin, with Weigert's elastic tissue stain and van Gieson's stain, and with the Unna-Pappenheim stain. A careful and thorough search was made for Aschoff bodies, Bracht and Waechter lesions and for any other evidences of inflammatory lesions. In the gross examination of the heart we carefully searched for macroscopic evidence of endocardial, pericardial and myocardial changes as well as for the commissural lesions of Lewis and Grant.[11, 12]

Table I shows the age period distribution of our specimens, the incidence of occurrence of vessels in at least some of the valves and the absence or presence of valvulitis.*

TABLE I

AGE PERIOD	NUMBER OF HEARTS INJECTED	NUMBER OF HEARTS WITH VESSELS IN SOME VALVES	NUMBER OF HEARTS WITH VALVULITIS
Fetal	3	2*	0
First decade	26	2**	0
Second decade	7	2	1
Third decade	9	4	2
Fourth decade	18	3	2
Fifth decade	10	3	2
Sixth decade	7	1	0
Seventh decade	4	0	0
Eighth decade	0	0	0
Ninth decade	1	0	0
Total	85	17	7

*Fetal vessels (seen microscopically only).

**Vessels only in base of aortic leaflet of mitral valve.

It will be seen that out of the eighty-five hearts studied, seventeen presented blood vessels in at least some of the valve leaflets. All the valves which did not show blood vessels on injection were, as stated before, examined microscopically as a routine and invariably failed to show blood vessels.

In Table II data are presented concerning the valvular leaflets which were vascularized, the extent of vascularization in the aortic leaflet of the mitral valve and the coexistence of valvulitis. Under the heading *extent of vasculature*, we have used the terms *incomplete* and *complete*. We shall explain these terms further on.

It will be seen that out of these seventeen hearts with valvular vessels, seven showed valves which were the seat of inflammation. Despite the presence of this inflammation we propose to present observations

*The term valvulitis will be used in this paper to signify a diseased valve of inflammatory origin. No effort will be made to indicate the specific nature of the valvulitis because this has no bearing on the anatomical observations to be made here.

TABLE II

SPECIMEN NUMBER	AGE	VALVES WITH VASCULAR INJECTION	EXTENT OF VASCULATURE IN AORTIC LEAFLET OF MITRAL VALVE	COEXISTENCE OF VALVULITIS
K-15	3 fetal mo.	mitral tricuspid	fetal*	none
K-20	6 fetal mo.	mitral tricuspid	fetal*	none
K-41	1 day	mitral (aortic flap)	incomplete	none
K-57	21 mo.	mitral (aortic flap)	incomplete	none
K-79	9 yr.	mitral tricuspid	complete	mitral tricuspid
K-54	18 yr.	mitral (aortic flap)	incomplete	none
K-4	20 yr.	mitral aortic	complete	mitral aortic
K-5	25 yr.	mitral	complete	mitral
K-7	27 yr.	mitral (aortic flap)	incomplete	none
K-35	29 yr.	tricuspid	incomplete	none
K-45	30 yr.	mitral	complete	mitral
K-74	31 yr.	mitral aortic tricuspid pulmonary	complete	mitral aortic tricuspid pulmonary
K-47	34 yr.	mitral (aortic flap)	incomplete	none
K-63	41 yr.	mitral aortic tricuspid	complete	mitral aortic tricuspid
K-70	44 yr.	mitral (aortic flap)	incomplete	none
K-32	45 yr.	mitral	complete	mitral
K-10	52 yr.	mitral (aortic flap)	incomplete	none

*Seen microscopically only.

concerning these valvular vessels which seem to us to indicate convincingly that the vessels are of noninflammatory nature.

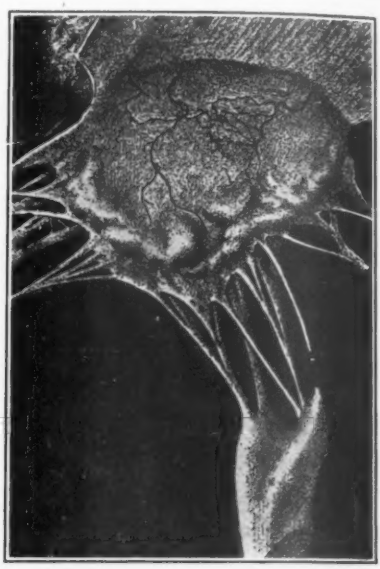
The aortic leaflet of the mitral valve, as said before, possesses more frequently and with greater constancy a rather characteristic vascular structure and we shall therefore concern ourselves with its description first. We propose first to describe what we shall name for purposes of brevity a *complete* vasculature, that is to say, a blood vessel structure which shows grossly, little if any signs of regression, or, as may be held by some, complete development. Further on we shall present briefly descriptions and illustrations of *incomplete* vascular structures, or such as we consider, for reasons which we will later state, to have undergone partial regression.

Fig. 1 shows a comparison of the blood vessel structure shown by Darier and by those of more recent writers. It will be observed that Darier's illustrations could serve equally well for those more recently described. The structure consists usually of two very distinct vessels descending from the auriculoventricular ring for a somewhat variable distance toward the valve edge. These are united by an arcuate vessel. One often finds a central branch somewhat more irregular in point of origin descending to join the arcuate vessel.

Fig. 2 shows a microscopic section of a valve from one of the hearts

studied by us. The section is stained with Weigert's elastic tissue stain and van Gieson's stain. It shows the presence of distinct arteries possessing an intima, an internal elastic membrane and a thick muscular media. The veins are also seen in the same plate possessing a muscu-

Postinflammatory vessels. (After Darier.)



Healed endocarditis. Postinflammatory vessels. (After Darier.)

Normal heart valve. (After Bayne-Jones.)

Subacute bacterial endocarditis. (After Gross.)

Fig. 1.—Comparison of vascular architecture in the aortic leaflet of the mitral valve.

laris but of a much more delicate structure than in the arteries. It is to be noted that what we describe as a muscularis we have found to consist essentially of smooth muscle. This can have therefore nothing to do with fetal remains of cardiac musculature.

Every valve which showed blood vessels on injection presented a very similar histological appearance of its vascular structures. These microscopic observations alone render it incontestible that these blood vessels are not of granulation tissue origin for we have failed to find any evidence to show that granulation vessels can take the structure of arteries and veins. We would quote in this connection from Mallory's *Principles of Pathological Histology*. In speaking of the development of granulation tissue capillaries he states: "New blood vessels arise as capillaries and take their origin from capillaries. * * * Later many of these newly formed capillaries disappear. Others persist and

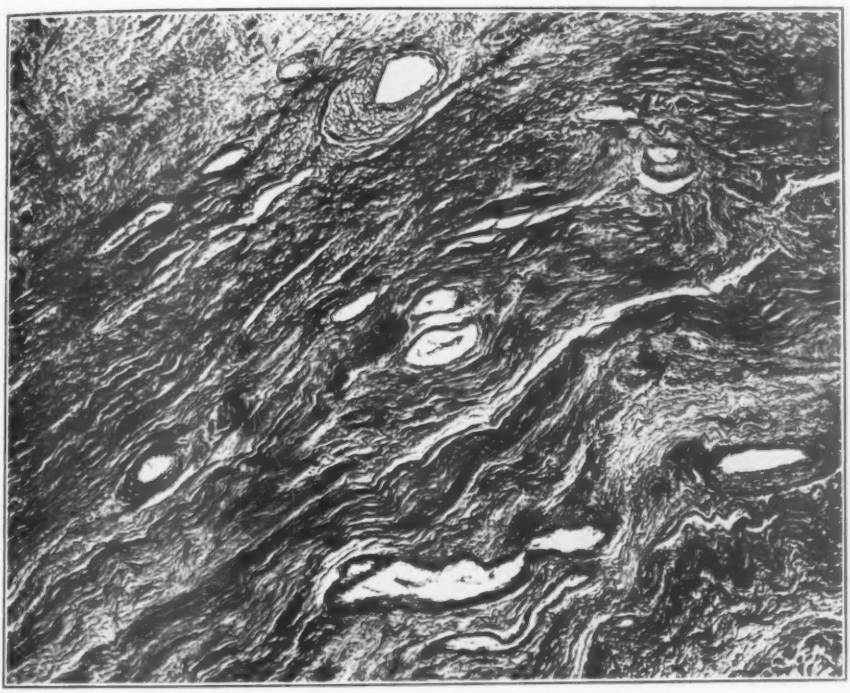


Fig. 2.—Photomicrograph showing *arteries* and *veins* in the aortic leaflet of the mitral valve.

may develop into larger blood vessels, but never into well-formed arteries and veins."

Fig. 3 shows the origin of these arteries directly from an anastomotic arch which joins the left and right coronary arteries. In some cases these Arteriae Valvulares spring from secondary arterial loops coming from the above-mentioned arterial arch. It will be observed that this is certainly not what would be expected if these vessels owed their origin to granulation tissue invasion. A strikingly similar architecture is presented by the vasculature in the normal aortic leaflet of the mitral valve of the calf. (Fig. 4.)

Fig. 5 shows the venous return from the mitral valve. This heart was injected through the coronary sinus and demonstrates a venous return structure which is strikingly different from that of the arterial. The essential characteristic of veins is seen in the profuseness of distribution and in the fact that they collect into a few larger thin-walled

Fig. 3.—Transparent specimen showing *complete* arterial arch in the aortic leaflet of the mitral valve of the human heart.

veins which empty directly into the *coronary sinus, with no direct communication with coronary arteries.*

We wish to reiterate at this point that not all valves from hearts other than those of the fetus possess blood vessels. We believe with Langer and Odinzow that regression takes place during fetal life, but as has been previously emphasized, there is a persistence in a small per-

centage of individuals. Those who have the persistence of blood vessels show an extraordinarily high incidence of valvulitis, if the valvular architecture remains *complete*. With this in mind let us now sum up the evidence which we have on hand to prove the existence of valvular blood vessels not of granulation tissue origin.

1. The constant and characteristic architecture of the blood vessels as opposed to the bizarre inconstant appearance of granulation tissue blood vessels.

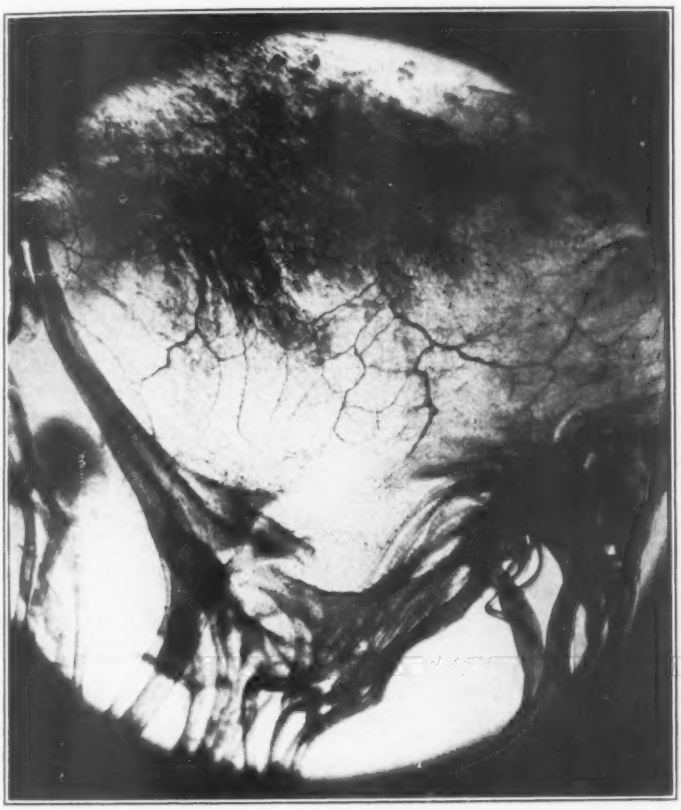


Fig. 4.—Arterial architecture in the normal aortic leaflet of the mitral valve of the calf.

2. The origin of the blood vessels from the anastomotic arch which springs from the main coronary arteries, and not from the neighboring capillaries as would be expected in granulation tissue.

3. The characteristic venous return from the valves into the coronary sinus.

4. The histological demonstration of arterial and venous structure of these vessels as shown by the possession in the former of an intima, internal elastica and a muscular media, and in the latter of the more delicate muscular wall.

On account of this constancy of structure, and because we feel we have proved that these vessels are not of inflammatory origin, we would suggest the following names for the arterial vessels in the aortic leaflet of the mitral valve.

The group of arteries as a whole should be called *Arteriae Valvulares.* That vessel which descends from the auriculoventricular ring nearer the anterior edge of the aortic cusp of the mitral valve (Fig. 3) we would name the *Ramus Anterior Cuspidis Mitralis.* That which descends nearer the posterior edge we would name the *Ramus Posterior Cuspidis Mitralis.* The arcuate vessel which joins these two, and from which a fringe of vessels descends toward the closing edge of the valve, we would name the *Ramus Arcuatus Mitralis.* As is seen in Fig. 3, this arch shows in some specimens a double convexity toward the edge of the valve. The less characteristic branch which is seen descending between the *Ramus Anterior* and *Ramus Posterior* to join the *Ramus Arcuatus* we would name *Ramus Intermedius Cuspidis Mitralis.* Very frequently anastomoses are seen to occur between these vessels and especially between the branches from the *Ramus Arcuatus.*

We do not propose here to go into detailed discussion on the rather frequent variations found in the distribution of these branches nor into the question as to what significance there is in the frequency with which valvulitis is found in the valves possessing a vascular structure in its *complete* form as described. We shall take up these questions in subsequent publications.

We wish merely to mention here that blood vessels are found with less frequency and with a less constant architecture in the posterior flap of the mitral valve; in all the other valves, and on rare occasions in the chordae tendinae passing from the papillary muscle into the closing edge of the auriculoventricular valves. This will also be taken up in a future statistical survey.

Thus far we have described what we call the *complete* vasculature of the aortic cusp of the mitral valve. It remains now to describe the vasculature as it is seen in this leaflet in the *incomplete* form (evidently in stages of arrested or incomplete regression or incomplete development).*†

Fig. 6 shows three stages of departure from the original typical *complete* vascular arch in the aortic leaflet of the mitral valve. Division "a" shows the absence of the *Ramus Arcuatus and Intermedius,* as well as partial regression of the *Ramus Posterior.* Division "b" represents a less complete form where only the first portions of the *Ramus Anterior* and *Posterior* are present. Division "c" shows a persistence of only the first portion of the *Ramus Anterior.* We have seen several stages in structure which are intermediate between these

*Dr. R. G. Hussey suggests that the presence or absence of vessels in the valves of the heart as described by us is a phenomenon of senescence. It seems reasonable to assume that the persistence of blood vessels in these valves may be accounted for in a way similar to the persistence of other structures in the body which we know do disappear at different periods of the life span.

†Although of the belief that blood vessels in valves are postinflammatory, Ribbert (Handbuch der Speziellen Pathologie und Histologie, Bd. 2, Herz und Gefässe, Springler, Berlin, 1924, p. 262) describes structures in the aortic flap of the mitral valve which closely resemble the *complete* and *incomplete* arch.

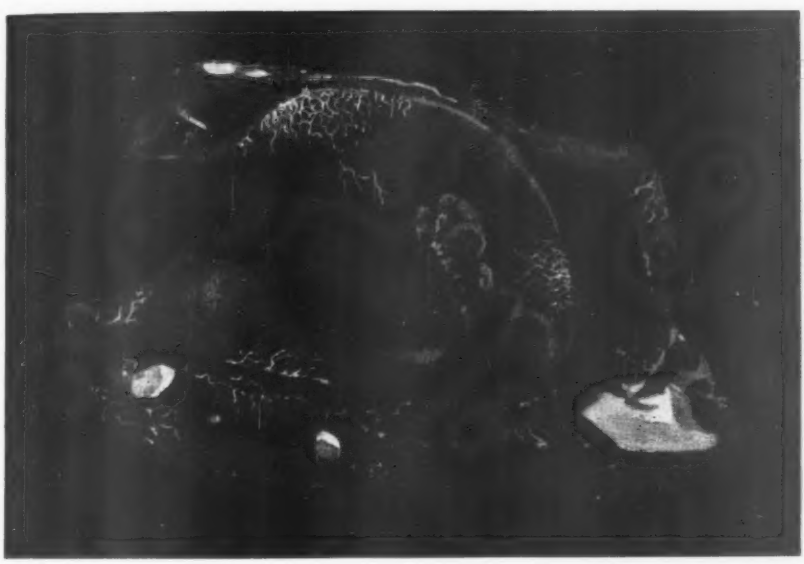


Fig. 5.—Painting from a "button-hole" mitral valve from a heart injected with barium sulphate gelatine (white) through the coronary sinus and showing the venous return network.

three grades of persistence of the vessels. It may be pointed out that even the remnants of valvular vasculature as briefly illustrated here in *incomplete* form occur at the characteristic site of the *complete* vascular arch and therefore furnish strong additional evidence of the preinflammatory existence of valvular vessels.

Since this paper is concerned only with the establishment of the fact that blood vessels of noninflammatory origin exist in a small percentage of hearts other than those of the fetus, we shall not discuss here the interesting speculations which our observations suggest on the significance of the almost invariable coexistence of the *complete* vasculature with valvulitis and of the less frequent occurrence of an inflammatory lesion in the *incomplete* vascular types.

We wish to conclude with the statement that we do not deny the occurrence of granulation tissue capillaries arising in the leaflets of

a. *b.* *c.*

Fig. 6.—*Incomplete* arterial arch in the aortic leaflet of the mitral valve. Three grades in the regression of arterial architecture.

the valve but insist that these invariably spring from capillary distributions of the vessels of the valves which are themselves of indisputable noninflammatory origin.

CONCLUSIONS

We believe that sufficient evidence has been presented in this paper to prove that blood vessels exist in some valves in a small percentage of hearts other than those of fetuses, that these are of developmental and not of inflammatory origin, that they occur most frequently in the aortic leaflet of the mitral valve and that they may exist either in what we call a *complete* or *incomplete* form.

On account of the constancy of architecture shown by these blood vessels, we have suggested names for the more characteristic branches.

The proof of the existence of *Arteriae Valvulares* should put new impetus into the study of the mechanism of valvular endocarditis and

render tenable the belief in the possible embolic origin of at least some forms of valvular endocarditis.

It is a pleasure to acknowledge our thanks to Professor M. C. Winternitz, Drs. F. S. Mandlebaum, E. Libman, and R. G. Hussey for criticism and advice. We are indebted to the King's County Hospital, Brooklyn, New York Hospital, and Harlem Hospital for anatomical material. To Professor Horst Oertel for his kindness in sending excellent injected material we also desire to express our gratitude.

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SPONTANEOUS RUPTURE OF THE HEART*

An Analysis of Fourteen Cases

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SPONTANEOUS rupture of the heart, although a terminal event and practically never diagnosed clinically, is of interest to the clinician in view of its relationship to a comparatively common lesion, namely, coronary sclerosis. However, in so far as the mode of exit is such that the patients are hardly ever seen by a physician before death or, at most, only when *in extremis,* it is of particular import to the pathologist and coroner or medical examiner. Almost every day the newspapers refer to sudden death in such terms as "acute indigestion," "apoplexy," "heart failure" and "angina pectoris." One may readily wonder if many of these are not due to rupture of the heart. Ten of the fourteen cases here recorded were medical examiner's cases, although one of the ten had been in the hospital a few days prior to death. It is indeed likely that if more cases of sudden death came to necropsy, the incidence of cardiac rupture would be greater than is now believed. Although the fourteen cases here reported were all subjected to autopsy at Bellevue Hospital, only five were from the hospital wards, the remaining nine having been found dead elsewhere—in homes, rooming houses, comfort stations and the like.

INCIDENCE

The published reports of Dr. Charles Norris,[1] Chief Medical Examiner of the City of New York, which include records of the five boroughs of the city, show a total of twelve cases of spontaneous rupture of the heart between January 1, 1922, and January 1, 1925. Ten of these twelve cases are described in this paper, together with four Bellevue Hospital cases. At Bellevue Hospital 2,690 autopsies were performed in the same period, 1,165 of which were medical examiner's cases and 1,525 hospital cases. Five ruptured hearts were found among these 2,690 autopsies. One of the five was a medical examiner's case, leaving a total of four cases of cardiac rupture among 1,525 hospital cases, or one to every 382 postmortems. Since the inauguration of the medical examiner's system in January, 1918, sixteen cases of spontaneous rupture of the heart have been recorded among 13,534 necropsies, an average of one to every 846 postmortems. Twelve of these sixteen oc-

*From the Pathological Laboratory, Bellevue Hospital, Dr. Douglas Symmers, Director; and the Office of the Chief Medical Examiner of the City of New York, Dr. Charles Norris, Chief Medical Examiner.

curred during the years 1922, 1923, and 1924, during which years 7,090 autopsies were performed, or one cardiac rupture to 590 necropsies. The other four occurred in 1920, during which year 1,688 postmortems were performed, an average of one ruptured heart to every 422 cases. None occurred during 1918, 1919 or 1921.* Hence, from a total of 15,059 necropsies, there have been collected twenty cases of spontaneous rupture of the heart, an average of one to every 753 autopsies.

Despite the comparative frequency of the lesion, most of the textbooks and monographs on medicine, and even books on heart disease, either do not mention it or dismiss it with a few lines, and these not infrequently are quoted from some authority of several decades ago. Often the statements quoted are misleading. As a result of this, the practitioner of medicine is apt to assume that spontaneous rupture of the heart is an extraordinarily rare event and to place it among the curiosities of medicine.

DEFINITION

As indicated in the title, only those cases are recorded in this paper that occurred spontaneously, that is to say, all cases of rupture of the heart due to extraneous causes have been excluded.

Anatomists have always disagreed as to the boundary line between the heart and the aorta. For purposes of the present discussion, the base line of the aortic cusps has been employed as the upper boundary of the heart. Therefore, rupture involving any structure above this line has been excluded. This is mentioned specifically because cases have been reported in the literature as "ruptured hearts" in which rupture occurred in the aortic sinus. Not infrequently cases of the latter type present themselves at autopsy at Bellevue Hospital, but they are always classified as ruptured aortae, both in the postmortem records of Bellevue Hospital and of the Medical Examiner's Office.

Another qualification necessary to come within the type of rupture here described is that the perforation and subsequent hemorrhage must have taken place into the pericardial sac. Hence, all cases of rupture of the valves, papillary muscles and interventricular septum are excluded.

HISTORY

It is not known whether the physicians of ancient times ever observed spontaneous rupture of the heart. A careful search fails to reveal any evidence to that effect. The condition is said to have been recognized during the seventeenth century by Harvey and Morgagni, both of whom described several cases in their writings. Morgagni himself died of rupture of the heart.

*Since this paper was written, two additional cases of spontaneous rupture of the heart have been encountered in the postmortem rooms of Bellevue Hospital.

In the early period of the eighteenth century several French writers, among them Morand and Portal, studied the condition in its relationship to etiology and symptomatology. During the nineteenth century, many authors brought forth monographs on the lesion. Dunlap, an Englishman, in 1866 collected twenty-nine cases from the literature. Quain,[2] in 1872, collected one hundred cases. Barth,[3] the French pathologist, who reported twenty-four instances of rupture in 1871, was the first to state the supposition that in the great majority of cases the main cause of the rupture was to be found in the coronary system. In 1885, Robin[4] published a work on the symptomatology of rupture. Meyer,[5] a German, reported in 1888 a detailed analysis of nine cases of his own, together with thirty-four that he collected from the literature over a period of fifteen years. In the latter half of the nineteenth century the lesion was described in the monographs of Rokitansky, Ziegler, Orth, Stokes, Weigert and others. Modern monographs and textbooks of medicine and pathology give little, if any, space to cardiac rupture.

ETIOLOGY

The present paper is based upon an analysis of fourteen cases of spontaneous rupture of the heart that were investigated by necropsy at Bellevue Hospital, five of the cases having been observed.

Although many famous men of medicine, notably John Hunter, Charcot, Nothnagel and William Pepper, died of coronary disease, which is usually the direct cause of cardiac rupture, and although coronary disease is considered more prevalent among professional men, yet postmortem statistics show that all classes of individuals are represented among the victims of heart rupture. Among the fourteen cases here reported, nine (60 per cent) were known to have been laborers; one had no occupation, but probably had been a laborer; the occupation of another man was unknown, but here too the probability pointed toward his having been a member of the laboring class; the only other male had been an embosser. In this series of fourteen cases there was not a single representative of a profession. However, as Bellevue Hospital is a public institution the great majority of patients admitted are of the poorer class, many of them laborers, and the postmortem material is necessarily drawn from this class. On the other hand, the Medical Examiner's cases are from all walks of life; yet there were no instances of rupture in a member of any profession among these cases.

The condition is not limited to the male sex, for two of the fourteen cases (15 per cent) were females—one a housewife, the other a schoolteacher. In Quain's series of one hundred cases, the sexes were equally divided. In this series there was marked dominance (85 per cent) of the male sex.

All but one of the cases were over fifty years of age, the exception

being the forty-three-year-old embosser. The oldest individual was a man of eighty-one. Of the eleven cases whose age was definitely known, seven were sixty years of age or over; of these, four were between sixty and seventy, two between seventy and eighty, and one was over eighty. The highest incidence, therefore, occurred in the seventh decade of life. In three of the Medical Examiner's cases, the age was unknown, but the approximate age given for all three was sixty years. The average age in the entire series was sixty-three years. This is in accord with the findings of most other observers. In Quain's cases, 63 of 88 ruptures occurred in persons over sixty years of age; of these, 33 were between sixty and seventy, 24 between seventy and eighty, and 6 over eighty years of age. The youngest case of rupture ever reported was that of Schaps[6] in an infant of four months; the rupture was associated with an embolic infarct of the left ventricle. Anderson[7] reported a rupture of the heart in a five-year-old child with "congenital stenosis of the coronary arteries" and "inherited syphilis."

Four of the fourteen subjects were born in the United States, three were of Irish nativity, two were German, one a Swede and four were of unknown nativity; of the latter, three possessed the features of the Anglo-Saxon race, the fourth of the Slavic race. We can draw no conclusions from these figures other than that apparently all races are represented among the victims of cardiac rupture.

It is impossible to say whether or not any relationship existed between hypertension and rupture of the heart in this series, since only a few of the patients had had blood pressure readings. One patient (Case I) had a pressure of 140/90 three days prior to death; another (Case III) had a pressure of 170/120 on the day of death; a third (Case IX), whose pressure three weeks before death was 190/110, gave a definite history and symptoms of an abnormally high blood pressure. Two years prior to her admission to Bellevue Hospital, this patient had been told that her blood pressure was abnormally high; she complained of frequent severe headaches, blurred vision and attacks of dizziness, and she developed a cerebral hemorrhage, for which ailment she was brought to the hospital. In this particular patient, apparently, and also in Case III, hypertension played the major rôle in the cause of heart rupture.

Physical exertion, no doubt, is the immediate cause of many cases of spontaneous rupture of the heart. However, some patients have been known to die of rupture while asleep. Rupture usually occurs during some physical effort, as, for example, straining at stool, lifting a heavy weight, or during a severe coughing spasm. In Cases VIII and XIV both victims were found dead sitting on toilet seats. In Case IX the subject was found dead in bed a short time after having been on a bedpan, the act of defecation most probably having been the exciting cause of rupture. Morgagni reported two cases of rupture during coitus.

George II of England died of a ruptured heart following defecation, falling down the palace stairway after having left his toilet. In Case XIII the victim was found dead at the top of a stairway after having ascended four flights; apparently this effort was the direct cause of rupture. In Case I the subject died by strangling on a morsel of food; the undue strain accompanying this act was, no doubt, the immediate cause of rupture. On the other hand, in Case II the patient was found dead in bed, apparently having succumbed while asleep. Extreme mental effort or psychic disturbance, although formerly thought to have been the cause of many ruptured hearts (an example of this being Philip II of Spain who was said to have died after hearing of the defeat of his armies at Piazenza), probably plays a minor part in the causation of this lesion. Some of the older authors described cases in which normal hearts had ruptured as a result of physical or psychic effort. This is now regarded as impossible; the gross pathological changes in the hearts must have been overlooked and, without microscopic aid, it was impossible to discover the causative lesion. It is interesting to note that Morgagni in the seventeenth century held that a normal heart muscle never ruptures.

With the possible exception of one patient (Case XI), whose father had died of "fatty degeneration of the heart" at the age of seventy years, heredity apparently was not a factor in the etiology of the lesion in the series herein recorded. Whether heredity plays a rôle in the etiology of cardiac rupture is questionable.

A history of the intemperate use of alcohol was elicited in four of the fourteen cases here described.

Six of the fourteen patients were obese; two of these were females.

No history of a previous acute infectious disease was obtainable in any of the cases. Although two of the fourteen patients stated that they had had syphilis, evidence of syphilis was found in neither at necropsy. A Wassermann test performed in one case (IX) was negative. Syphilis as a causative factor played no rôle in this series of cases. Only two (II and VII) showed signs of syphilis at necropsy, one case presenting syphilitic aortitis and interstitial orchitis, the other syphilitic aortitis. Neither of these two described lesions had any influence on the lesion underlying the ruptures.

Contrary to statements appearing in medical textbooks and periodicals, syphilis and, more specifically, gumma are rarely the cause of spontaneous rupture of the heart. At Bellevue Hospital, among 10,200 autopsies, only one case of rupture of the heart due to syphilis was discovered. This particular case of ruptured gumma, which, incidentally, was a coroner's case of 1916, was reported by St. George[8] because of the extreme rarity of this type of lesion. Not a single instance of ruptured gumma of the heart is to be found in the records of the Office of the Chief Medical Examiner of the City of New York since its

establishment in 1918, although more than 13,000 autopsies had been performed since that year. Yet, in *Progressive Medicine* for September, 1921 (p. 119), there appeared the following statement: "* * * It may be noted that sudden death from rupture of the heart is usually due to gumma of the myocardium." In further refutation of this statement and many others of similar tone, I may refer to the nineteen cases of thrombosis of the coronary arteries with infarction of the heart reported by Wearn,[9] among which he was unable to find any evidence of syphilis and concluded, therefore, that syphilis was not an etiological factor in his series. Incidentally, MacCallum[10] states that gummata are rare in the heart.

In view of the findings in these fourteen cases of spontaneous rupture of the heart and the postmortem statistics of Bellevue Hospital and the Medical Examiner's Office of the City of New York, I am forced to conclude that syphilis as a cause of cardiac rupture is a rarity. However, if, as Osler[11] states, syphilis is one of the most important single causes of arteriosclerosis, then syphilis as an indirect cause of rupture is, perhaps, more frequent. In this connection, it is to be recalled that modern pathologists recognize a sharp distinction between arteriosclerosis, which is purely a degenerative lesion, and syphilitic aortitis, which is an inflammatory disease, and that syphilis as a contributing factor in the production of arteriosclerosis is probably negligible.

Arteriosclerosis, particularly sclerosis of the coronary arteries, was the predominant etiological factor in all of the fourteen cases in this series. Coronary occlusion, which includes thrombosis, embolism and atheromatosis, with subsequent infarction and necrosis of the heart muscle, although the immediate cause of rupture in all the cases here described, is really a process secondary to coronary sclerosis. In turn, when searching for a cause for arteriosclerosis, one becomes entangled in a maze of considerations, namely, hypertension, chronic intoxication, overeating, renal disease, "the stress and strain of modern life" and, finally, the involution process accompanying old age.

Localized changes in the myocardium as a result of coronary obliteration are the usual direct cause of spontaneous rupture of the heart. These changes consist of infarction of the heart wall with subsequent necrosis. At this point, it may be of interest to note that rupture may occur long after the original infarction of the heart wall. In fact, Krumbhaar[12] believes that the infarcted area rarely ruptures while in the stage of softening, but remains as a vulnerable spot even after the necrotic area has become organized. The findings in the cases here described, however, do not substantiate this statement, for in nine cases (65 per cent) the rupture occurred during the stage of acute softening. Sudden or gradual distention into an aneurysm may take place (acute and chronic aneurysm). Rupture of these cardiac aneurysms has been reported not infrequently. However, Elliott[13] says that chronic cases

of cardiac aneurysm seldom terminate in rupture, but usually by progressive myocardial failure. Legg[14] found rupture in only 7.7 per cent of 90 reported cases. As Wooley[15] states, in some cases, because of limitation of the dilatation by pericardial adhesions, rupture does not take place and the bulging becomes permanent.

Among the rarer causes of spontaneous rupture of the heart, are endocardial ulcerations accompanying endocarditis, with subsequent perforation of the heart wall. Claytor[16] recently reported a case of this type due to the Streptococcus viridans. Another recognized cause of rupture is embolism of a coronary artery due to vegetative endocarditis, in which an embolus from a valve suddenly plugs the mouth or lumen of a coronary vessel. Cases of this type are usually found in young adults. Abscess of the myocardium associated with or following general septic infection has been known to cause rupture of the heart. Hammer[17] described a case of the latter type and incidentally stated that he was able to find only five other instances in the literature. Mills[18] cites Graham as having reported a case of hydatid cyst in the left auricle of the heart in a man who dropped dead while at work, as a result of the cyst rupturing through the wall into the pericardium.

Tubercles and gummata of the myocardium are also among the rarer causes of cardiac rupture. Anderson[7] reported a case of rupture in a five-year-old child as a result of congenital syphilis with stenosis of the coronary arteries. Congenital stenosis of the isthmus of the aorta is said to have been the cause of rupture in a few instances. Rupture of an aneurysm of a coronary artery was formerly held to be a not infrequent cause, but this is now considered doubtful. Carcinomatous and sarcomatous metastases in the heart wall have been listed among the rare causes of spontaneous rupture of the heart.

SYMPTOMS

The onset of rupture of the heart is sudden and death is usually instantaneous, that is to say, within a few minutes. However, it is important from the medicolegal point of view to remember that death is not always instantaneous. Several cases have been reported in which death did not occur for hours or even days. Engelhardt[19] observed a man with a ruptured heart for twelve hours. The case of a patient who lived twenty-four hours after rupture is reported by Curtin.[20] Osler quotes a case in which the patient walked up a steep hill after the onset of symptoms and lived for thirteen hours after rupture. He also states that a case is on record in which the patient lived for eleven days. Quain reported one patient who lived eight days, one six days, another three days and five over forty-eight hours. Possibly some of these patients were still in the stage of infarction.

Of the five cases here presented that were observed in the hospital wards, three died immediately after rupture took place. The other

two (Cases IV and IX) lived an hour or two after the presumed time of rupture. It is impossible to say how long the remaining nine patients lived after the accident, in view of lack of data.

In those cases in which death does not occur for hours or days after the rupture, there are several factors present. The rupture itself is sometimes barely large enough to admit a probe (as in Case VII). In other instances it is many centimeters in length (as in Case XIII). (See Fig. 1.) It is likely that the rapidity of death is dependent not alone on the size of the rupture but also on the seat of the opening and the manner of tearing of the heart wall.

Cessation of the heart beat does not necessarily follow rupture, for in many of the recorded cases the patients have lived for periods varying from several hours to days after the presumed time of rupture. Death may result from shock or from tamponade of the heart, due to overdistention of the pericardium with blood. The latter is usually considered the cause of death by most authorities. In most cases reported, cerebral anemia due to acute hemorrhage cannot be considered the cause of death because the amount of blood found in the pericardial sac, as a rule, is not sufficient. The average quantity of blood removed in this series was 318 grams—the smallest 200 grams, the largest 600 grams (Case XIII). Hence, loss of blood alone does not explain the cause of death. Stokes[21] was opposed to the theory that death was due to compression of the heart as a result of increased pressure in the pericardial sac. He stated that the heart in cases of pericarditis with effusion could stand considerable increase in pressure. However, as Meyer states, this condition is not comparable to rupture of the heart, since in one the increase is gradual and in the latter sudden. Some authors suggest that there may be some unknown reflex function which enters into the question of the mechanism of death.

As previously mentioned, rupture frequently occurs at the moment of some physical effort, at which time sudden and marked strain is made upon the weakened cardiac wall. In five of the cases (36 per cent) here presented the patients died at the time of exertion. On the other hand, rupture may occur during sleep, as in one of the cases (II) of this series and as reported by others.

The final attack is sometimes preceded by symptoms referable to the heart, but in many of the cases no such symptoms are mentioned. None of the five Bellevue Hospital patients gave an antecedent history of symptoms referable to the cardiovascular system for any appreciable length of time prior to death. Three, including a forty-three-year-old man (Case III), gave a cardiac history of two weeks' duration; another a history of occasional attacks of palpitation of the heart and precordial pain during the two years prior to death; but these were secondary complaints, hemiplegia being the principal one. In the fifth patient the duration of symptoms was not known, but, while in the

hospital, he apparently showed no signs of cardiac disturbance; he was up and about the ward until the onset of an attack of excruciating precordial pain about an hour before death. In none of the other cases of this series was there an antecedent history of cardiac disturbance, with the exception of one patient (Case VI), who complained of severe substernal pain four days prior and up to the time of death. Hence, it is possible for a person to be in apparently normal health one minute and in the next be afflicted with the unrelenting pain of coronary occlusion, followed by death due to rupture of the heart.

With the exception of four cases (I, III, VI, IX), the symptoms of all those patients from whom it was possible to acquire a history did not lead one to postulate any real involvement of the coronary circulation. In Case III, the attacks of sudden excruciating and persistent pain was the outstanding complaint; the pain was precordial and radiated to the shoulders and left forearm; nitrites and morphine did not relieve it. In all, this patient had seven of these attacks of "angina pectoris" during the ten days prior to admission to the hospital. His final attack, which began on the night before admission as an agonizing precordial pain without remission until death intervened (twenty-four hours after onset), was typical of coronary occlusion. As Reznikoff[22] stated in his report of this case, the sudden collapse, projectile vomiting and immediate death were probably coincident with rupture of the heart. Substantiation of the fact that death was almost instantaneous after rupture in this case rests on Reznikoff having heard the extraordinary sound over the precordium during the patient's death agony (see case report). I make special mention of this because some of the older authors, particularly those of the French school (Barth and Robin) were convinced by bedside observation of patients with presumed cardiac rupture, that after the initial attack of precordial pain every subsequent attack was due to further tearing of the muscle tissue. If, as Meyer suggests, their observations were correctly interpreted, there must have been many patients who, after cardiac rupture, lived more than a week. The interpretation in question, however, is not accepted by modern investigators.

Since rupture *per se* is dependent on disproportion between intracardiac pressure and the resistance of the weakened heart well, it is more likely to occur during systole, when the pressure is greatest. Apparently there are instances where a single systole seems sufficient to begin and complete the rupture. These are usually the cases where, at necropsy, the tear is large and comparatively regular in outline (Case XIII, see Fig. 1). In these cases death is instantaneous. On the other hand, there are patients in whom life is prolonged minutes or hours and in whom the tear is small and irregular, so that the blood seeps through the heart wall. Cases V, VII, IX, and XII (see Fig. 2) were probably of this type.

Sudden excruciating and persistent pain, with or without radiation, usually substernal or precordial in location, but which may also be epigastric, is commonly the first and predominant symptom. Extreme dyspnea or orthopnea, cyanosis and restlessness accompany this pain as a rule. Following these, the patient presents the picture of shock,

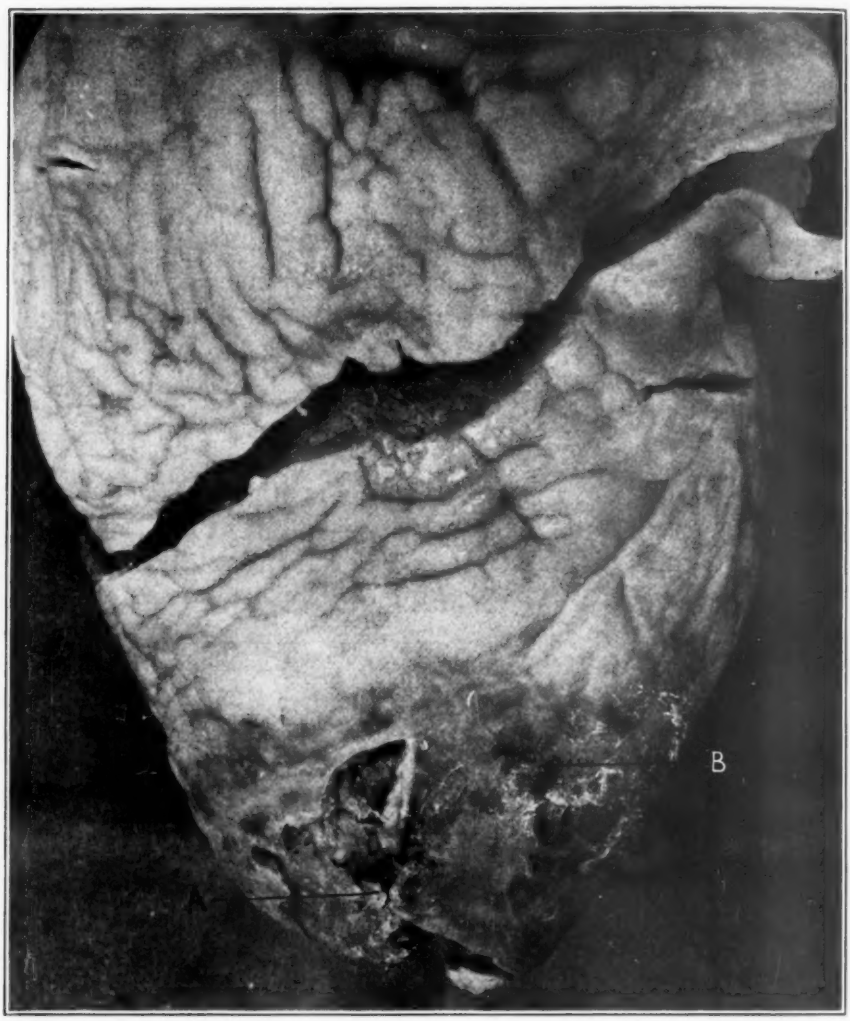

Fig. 1.—Case XIII. Anterolateral view of heart. Note the marked enlargement of the heart. *A*, site of large, ragged rupture; *B*, fibrinous pericardial exudate.

namely, pallor, cold and clammy skin and pinched facial expression. However, as previously mentioned, death may be postponed for minutes or hours. Nevertheless, the onset is the same, but some of the secondary symptoms, i.e., dyspnea, cyanosis, etc., may disappear. In addition to the above-mentioned symptoms, some patients present those of gastro-

intestinal disturbance, as typified in Cases III and VI, namely, nausea, vomiting and gaseous eructations. Case VI was diagnosed as "ptomaine poisoning" several days prior to death; she had thrombosis of a coronary artery at that time, as was subsequently proved at necropsy. This

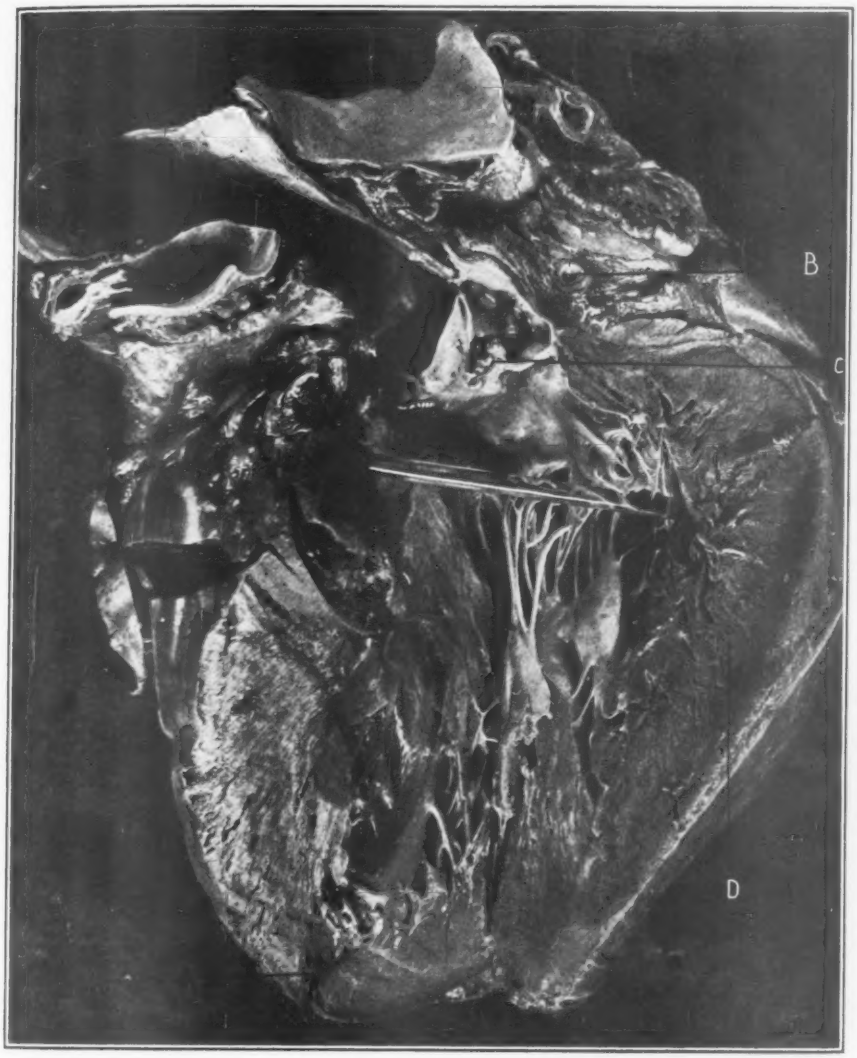

Fig. 2.—Case V. Interior of left ventricle: *A*, site of rupture; *B*, marked sclerosis of coronary vessel; *C*, calcification of aortic cusps; *D*, hypertrophy of ventricle wall.

is in accord with an observation made by Libman,[23] who noted that, if a patient gave a history of having had an attack that was diagnosed as "ptomaine poisoning" or "gastritis," and afterward had to stop walking because of pain in the chest, the patient most likely had thrombosis of a coronary artery.

When the pain is referred to the epigastrium or to the right upper quadrant of the abdomen and is accompanied by nausea and vomiting, and if the patient survive the initial insult, an acute intraabdominal lesion, such as acute pancreatitis, ruptured gall bladder or perforated peptic ulcer, may be suspected. The other symptoms and signs, e.g., spasm of the recti muscles, may serve to strengthen this suspicion unless properly interpreted. Failure to take into consideration the condition of the coronary arteries of these patients has resulted not infrequently in surgical intervention. Levine and Tranter[24] emphasized the close resemblance of cardiac infarction; the precursor of rupture, to acute abdominal conditions.

Examination of the individual at the time immediately following rupture may reveal the absence of heart sounds or the presence of feeble and distant sounds, or, as Reznikoff heard in Case III, a "continuous, muffled, rushing, rumble which is louder during expiration." On percussion, the area of cardiac dullness is increased and assumes a triangular outline, similar to that found in pericarditis with effusion. The pulse may be imperceptible, as in Case III, or small, feeble and rapid. The heart rhythm may be regular, as in most of the cases, or irregular as in Case III. A pericardial friction rub may or may not be heard at this time; as a rule not, because of hemopericardium. The temperature is subnormal during the period of shock, but an immediate rise occurs should the patient survive. During the stage of infarction prior to rupture, fever is usually present. In Wearn's cases of thrombosis with infarction, the average temperature was 101° to 102° F. A blood count taken at the time of rupture would probably reveal a leucocytosis. (This may be accounted for by the necrosis of the heart muscle which was found on microscopic examination in most of the cases here presented.) Only three of Wearn's nineteen cases of cardiac infarction did not have a leucocytosis.

A sudden and marked fall in systolic blood pressure occurs after rupture has taken place, but just prior to rupture there is a sudden rise in intracardiac pressure, which manifests itself as a general rise in blood pressure. Unfortunately for purposes of differentiation, a rapid and marked fall in pressure also occurs in coronary thrombosis, as has been shown by Allbutt[25] and others, and this is considered of distinct diagnostic value in the latter condition.

Electrocardiographic tracings taken at the time of rupture would probably show ventricular fibrillation. In Case III, in which the cardiogram (see Fig. 3) was taken a few hours prior to death, there was an alternating bradycardia and tachycardia. Electrocardiographic tracings taken during the time of rupture of the heart are not to be found described in the literature, although many readings have been reported in cases of coronary thrombosis, the precursor to most ruptures. Smith[26] found that changes in the T-deflection and a decrease

in the amplitude of the QRS complex were the most constant findings in his observations of coronary thrombosis. He states that the former is apparently due to an area of infarction in the apical region of the left ventricle, and that the latter is associated with extensive and disseminated changes in the myocardium. In Wearn's cases of thrombosis and infarction, disturbances of the T-wave and diminished amplitude were also the most constant findings. He states, however, that no one form of electrocardiogram is characteristic of the condition.

It is no doubt true that spontaneous rupture of the heart is usually undetected clinically. Given the above symptoms and signs, however, in a person over sixty years of age, even though the previous history may be negative, one ought to make a "reasonably certain diagnosis."

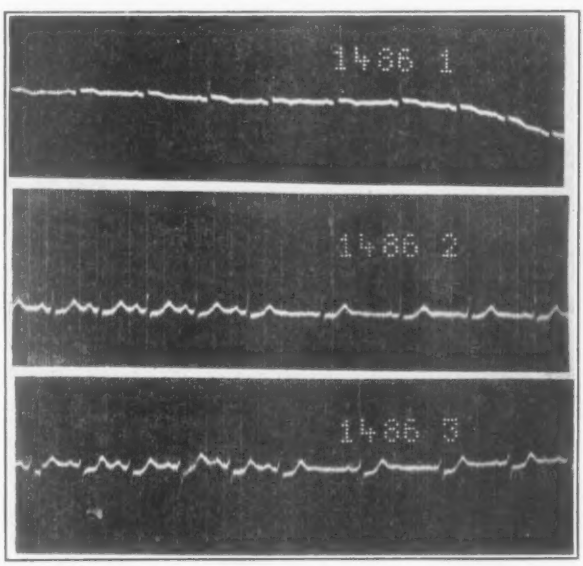

Fig. 3.—Case III. Electrocardiogram taken a few hours before death showing an alternating tachycardia and bradycardia.

NECROPSY FINDINGS

The outstanding feature found at necropsy in this series, and which accounts for the main lesion, i.e., rupture of the heart, is sclerosis of the coronary arteries. The coronary arteries were involved to a greater extent than any of the other vessels of the body. Possibly, as Smith[27] states, the coronaries owe their degeneration in some degree to the mechanical irritation to which they are subjected with each contraction of the heart. The degree of sclerosis varied in the different branches; the left coronary artery, particularly the ramus descendens of this vessel, was the artery which, as a rule, was involved to the greatest extent in the process of sclerosis. Among the fourteen cases, the most marked involvement in eleven of the hearts was of the left coronary artery or

its branches. The ramus descendens was involved in nine of these eleven cases; the main trunk and the circumflex branch of the left coronary in only one instance each. The right coronary artery was involved in two cases (VI and X). In one case the right and left coronary arteries were equally affected (XII).

The site of thrombosis in these cases coincides with the findings just given. A thrombus was found in twelve of the fourteen specimens. In the two cases in which no thrombus could be discovered, the coronaries were practically completely occluded by patches of calcification (X and XII). The vessel of election in eleven of the fourteen cases was the left coronary artery; of these, nine were in the ramus descendens, one in the main trunk and one in the circumflex branch. Given the above figures, it must be admitted, as Herrick[28] stated, that the reputation of the descending branch of the left coronary as the artery of sudden death is not undeserved. In only one instance was the right coronary artery thrombosed (Case VI, see Fig. 5), although in Case X, despite the apparent absence of a thrombus, the right coronary presented more marked sclerotic and calcific changes and greater constriction of its lumen than the left coronary. In contradistinction to the view of Gross,[29] it might be permissible to suggest that "a man is as old as his left coronary artery." Why the left coronary artery is most often the seat of thrombosis is not known. However, it is the vessel most often mentioned in describing the usual seat of coronary thrombosis. In sixteen of the nineteen cases of thrombosis reported by Wearn, the occlusion occurred in the descending branch of the left coronary artery. In one instance (Case VII), the heart presented complete occlusion by a recent thrombus of the circumflex branch of the left coronary artery and of the ramus descendens of the same vessel by an old thrombus. The area of rupture was supplied by the former vessel, the thinned fibrous area near the interventricular septum by the latter vessel. Case XI presented a picture directly the opposite of that above mentioned, the circumflex branch being occluded by an old and the descending branch by a recent thrombus. Both these cases (VII and XI) furnish direct evidence that coronary thrombosis in human beings is not always fatal. Aschoff, Krehl, Dock, Osler, Herrick and others have observed cases of this kind, and autopsy experience in Bellevue Hospital tends to verify the frequency of the condition. There can no longer be any doubt that anastomoses between the coronary arteries do exist, as so beautifully demonstrated recently by Gross[29] and previously proved by Janin and Merkel, Hirsch, Spalteholtz and others. The large areas of fibrosis (old infarcts) present in both of the hearts above referred to were certainly of long standing and were the result of earlier occlusion of the vessels of supply.

The localization of the rupture narrows itself down in all the cases of this series to the left ventricle. Investigators agree that the majority

of tears involve the left ventricle. Quain reported seventy-six ruptures of the left ventricle out of one hundred cases; Meyer twenty-five out of thirty-eight cases. The anterior surface was involved in ten of the

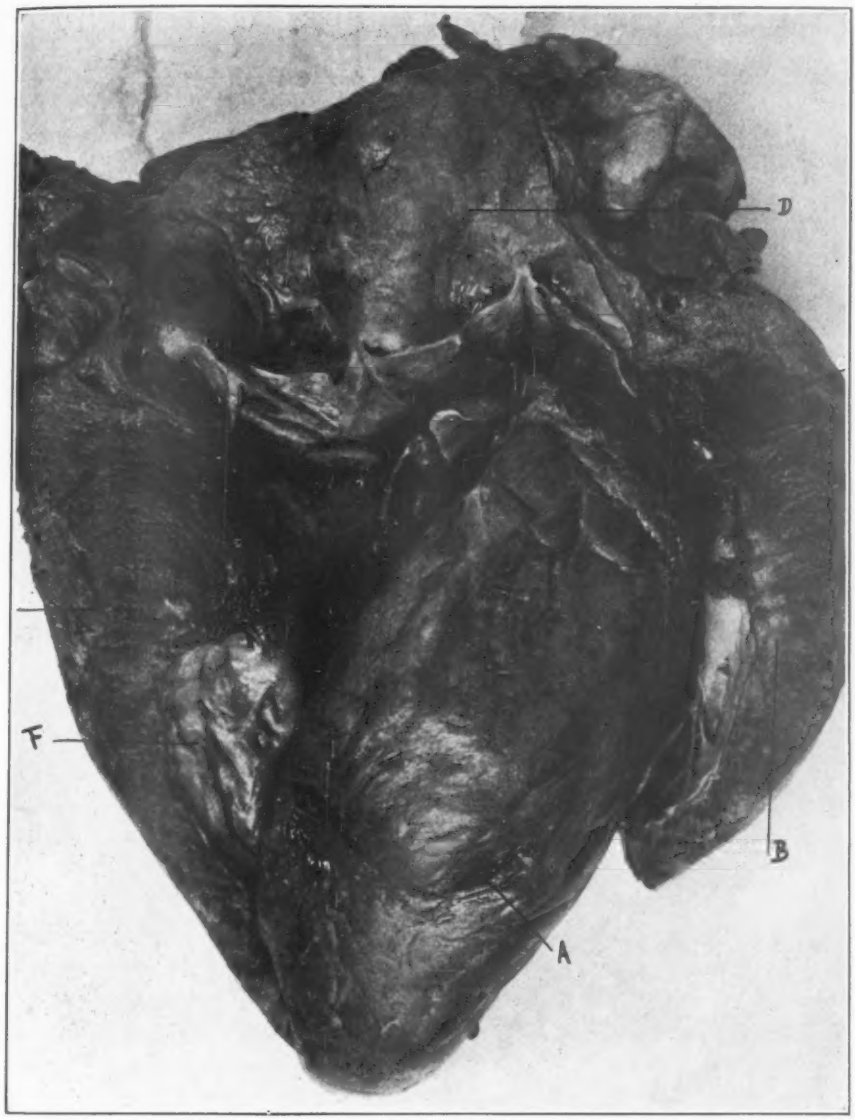

Fig. 4.—Case VII. Posterolateral view of heart, showing a part of the left ventricular chamber; *A*, site of rupture; *B*, fibrosis of heart muscle; *C*, recent thrombosis of circumplex branch of left coronary artery; *D*, syphilitic aorta; *E*, old calcified thrombus of the descending branch of left coronary artery; *F*, old infarct of left ventricle wall and interventricular septum.

present fourteen cases, the posterior surface in three instances, and both surfaces in one instance (Case XIII), where the tear was 7 cm. long and was situated at the apex of the heart. In Quain's series, forty-five of

the seventy-six cases of rupture of the left ventricle were in the anterior surface.

Although none of the cases here described presented rupture of the right ventricle or of the auricles, it has nevertheless been reported from time to time. Quain found thirteen ruptures of the right ventricle,

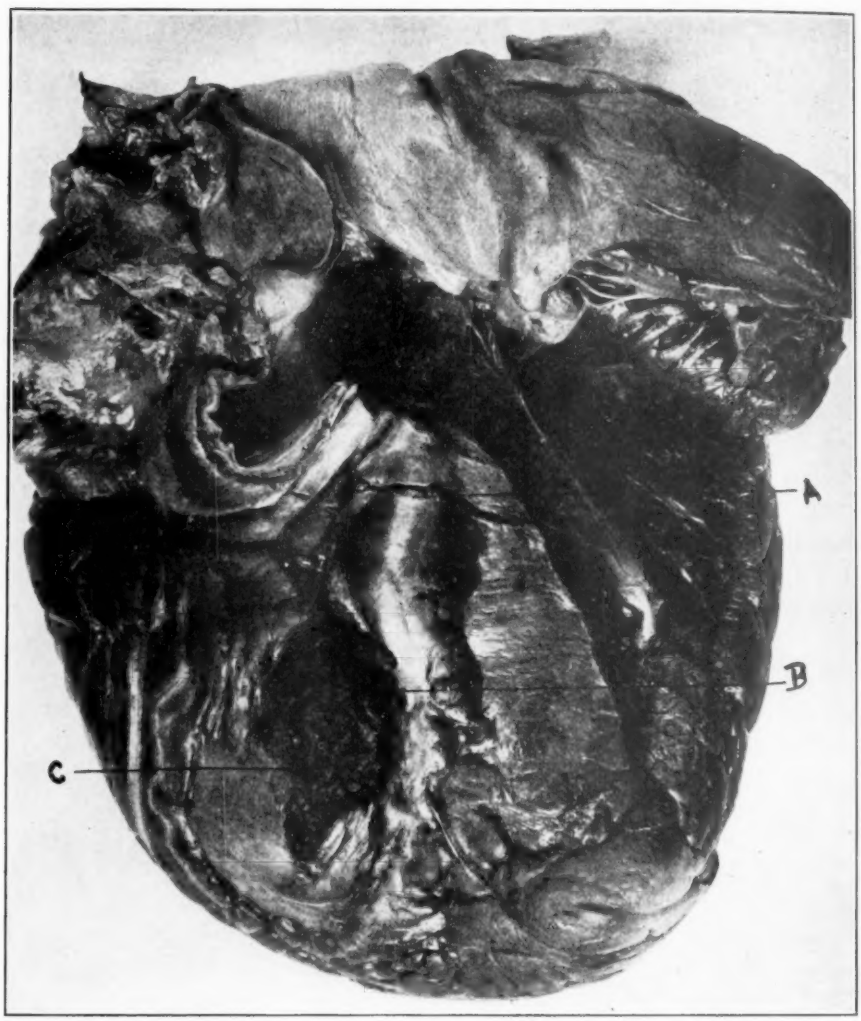

Fig. 5.—Case VI. Posterior surface of left ventricle; *A*, thrombosis of right coronary artery; *B*, site of rupture adjacent to the interventricular septum; *C*, heart muscle denuded of pericardium.

seven of the right auricle and two of the left auricle in his review of one hundred cases. Meyer collected seven ruptures of the right ventricle and four of the right auricle in thirty-eight cases reviewed.

The predilection for the anterior surface of the left ventricle is due to the distribution of the ramus descendens of the left coronary artery.

In the three cases here described where the tear occurred in the posterior surface of the left ventricle, the causative lesion was in the right coronary artery in two instances, and in both vessels in the third case.

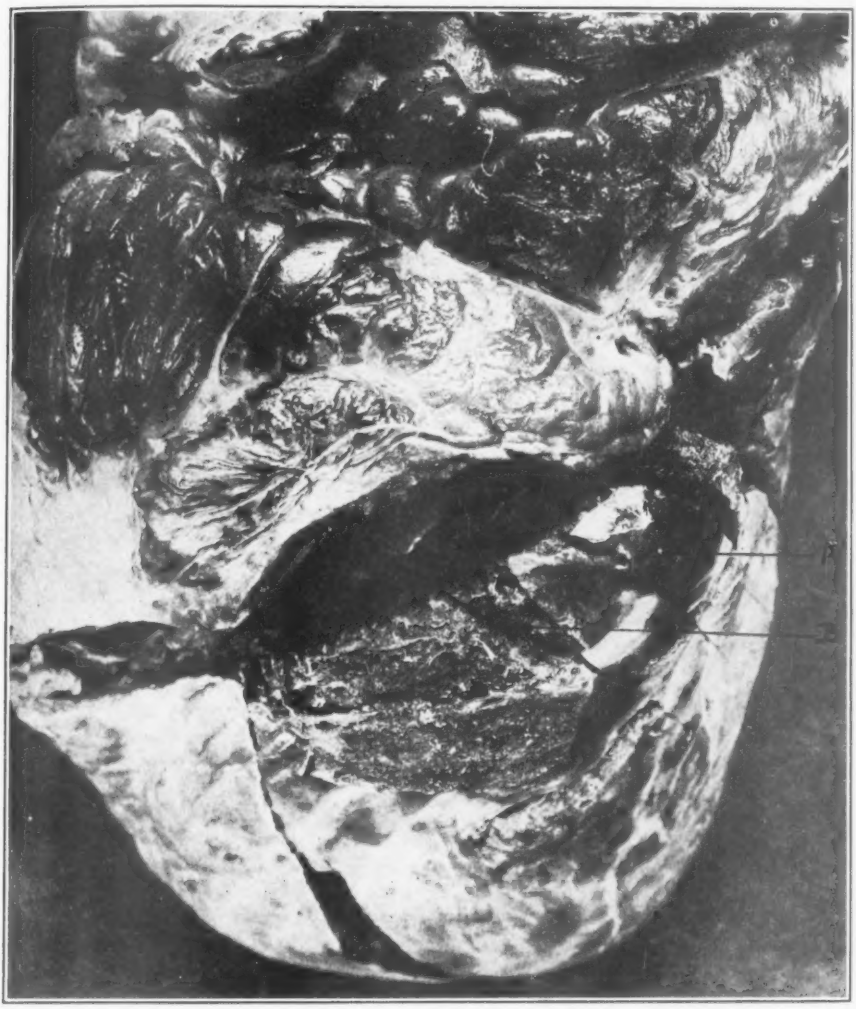

Fig. 6.—Case III. *A*, site of rupture; *B*, infiltration of subepicardial and muscle tissue with blood.

The apex of the left ventricle is named as the usual site of rupture. In this series, seven of the cases (50 per cent) presented rupture at this point, the other ruptures being situated about midway between the apex and base. Here again the selection of the apical region as the site of rupture is due to the distribution of the ramus descendens branch of the left coronary artery, although the thinness of the heart

wall in this region must be taken into account as a contributing factor.

The size of the rupture may vary from a few millimeters, as in Case I (8 mm.) to many centimeters in length, as in Case XIII (7 cm.). The average length of rupture in this series was 2.5 cm. Instances have been reported in which the tear involved the entire length of a ventricle wall. The laceration is usually larger on the external surface

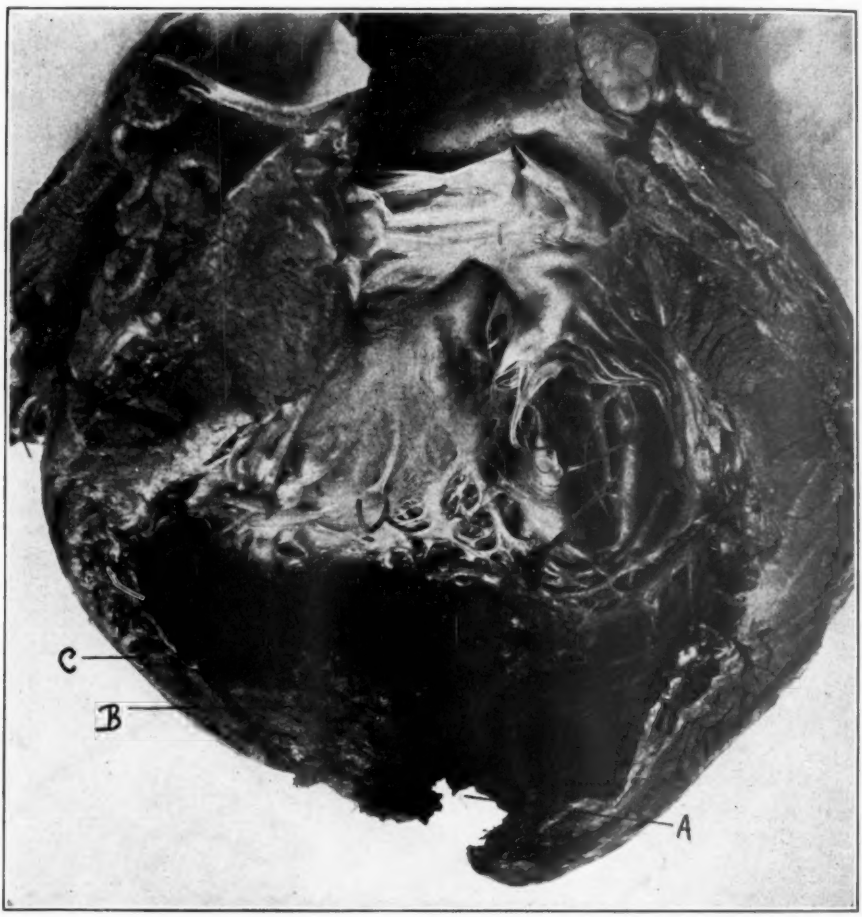

Fig. 7.—Case IV. Interior of left ventricle; *A*, site of rupture; *B*, firmly adherent thrombus; *C*, marked thinning of ventricular wall.

than on the internal, as exemplified in Cases I, V, VII, and X, but may be the opposite, as in Case XII. No characteristic appearance is assumed by the line of rupture. Some are ragged and irregular (Cases III, VI, XI, and XII); of these a few (Cases IV and XIII) resemble the traumatic ruptures of the heart found in crushing accidents, where the entire apex is torn. Others are smooth and regular in outline (Cases VIII and IX) and resemble incised wounds of the

heart, and, finally, there are those that take a tortuous course through the heart wall (Cases V and VII) with both a small external and internal opening. Ruptures are sometimes multiple. For example, Ley[30] described a case with three vertical openings; and as many as five perforations in one heart have been reported.[31] Rupture may be incomplete, involving only a portion of the heart wall. However, when this condition is found, care must be taken that one is not dealing with a ruptured coronary artery rather than rupture of the heart wall. Incidentally, this condition (coronary rupture) "apoplexia cordis," plays an important rôle in the etiology of ruptured hearts, according to statements in the older reports. Stokes was the first to state that coronary rupture was merely a result of involvement of the vessel at the time of tearing of the heart muscle. Hence "apoplexia cordis" as a cause of cardiac rupture is now considered negligible.

Parietal thrombi were found adherent to the inner side of the rupture in seven of the cases here reported (50 per cent). These are not an infrequent finding in cardiac rupture, especially in those cases reported as having occurred in "aneurysms" of the heart wall which have ruptured spontaneously. These thrombi may prevent rupture in some cases; in others, where rupture has occurred, they may prolong life, at least temporarily, by diminishing the escape of blood into the pericardial sac.

Pericarditis was present in four of the present series, and was confined to the area of rupture, presenting a fibrinous appearance. The formation of pericardial adhesions at the time of infarction may prevent rupture or, at least, prolong life after rupture. According to Gorham,[32] a pericardial friction rub is one of the most important diagnostic signs of cardiac infarction.

Aneurysmal dilatation of the heart wall was present in four of the cases; none were distinct aneurysms (see Figs. 1 and 7). Three of the four cases had pericardial adhesions surrounding the site of dilatation and also parietal thrombi, which occupied the dilated portion of the ventricular chamber. In three cases, rupture took place at the dome of the dilatation; in the fourth (Case XI) rupture occurred at the thickest portion of the left ventricle, despite the fact that the wall adjacent to the interventricular septum was only a few millimeters in thickness and was completely replaced by compact fibrous tissue.

The average weight of the hearts was 454 grams; the heaviest weighed 700 grams (Case XI), the lightest 320 grams. Enlargement was present in ten of the cases; the other four were normal in size.

In nine of the fourteen cases (65 per cent) the rupture occurred in the stage of acute infarction; in one case (V) (7 per cent) in the early stage of organization; in three cases (31 per cent) it occurred at the site of aneurysmal dilatation of the heart wall, where fibrous tissue predominated (Cases IV, IX, and XIII).

Some of the infarcted areas assumed the characteristics of an anemic infarction, but the majority were of the hemorrhagic type. All were irregular in outline and considerably less in extent than the region supplied by the occluded artery. The rupture was usually situated in the center of the softened area. Adjacent to the area there was much fibrous tissue replacement.

Many of the hearts presented small, pearly-white, fibrous areas scattered about the musculature. These were probably the result of previous obstruction or narrowing of smaller branches of the coronary vessels. Besides these small areas just described, Cases VII and XI also presented large, dense, white scars of irregular outline, involving a considerable portion of the thickness of the left ventricle wall (see Fig. 4). These were due to previous occlusion of the vessels of nutrition and were, no doubt, of long standing.

The valves were normal in four cases. There was slight fibrous thickening of the aortic cusps and mitral valves in four instances; the mitral valve alone in one instance. Sclerosis and calcification of the aortic cusps were found in two cases; in one of these fusion of two cusps had taken place. The remaining cases, including one with syphilitic aortitis (Case II) showed sclerosis of both aortic and mitral valves. The other case of syphilitic aortitis (VII) showed slight sclerosis of the mitral valve. In the only case in which a murmur was heard during life, no pathological change in any of the valves was found at necropsy, namely, Case XI, in which a blowing systolic murmur was heard at the mitral area.

None of the ruptures herein described involved any of the valves. Instances have been reported, however, in which valves have been torn during the process of cardiac rupture.

Gross examination of the aortae of these cases revealed the typical picture of arteriosclerosis in all but two cases, the exceptions being two cases of syphilitic aortitis (II and VII), in which, however, sclerosis was also present (see Fig. 4). In neither of these two cases were the orifices of the coronaries narrowed or occluded by the syphilitic condition in the aorta. Unfortunately, the coronaries of only one of the two cases were searched microscopically for signs of syphilitic involvement (Case VII), but the changes found were all due to arteriosclerosis. Hamman[33] states that syphilitic aortitis frequently narrows or occludes the mouths of the coronary arteries, but it rarely involves the coronaries themselves. In only two cases (I and VI) were the orifices constricted by the marked arteriosclerotic changes in the aorta. However, in both the site of thrombosis was approximately 3 cm. from the mouth of the vessel.

Microscopic Examination.—The microscopic picture presented in one coronary artery was practically the same for all of the cases examined (twelve), namely, marked sclerosis with calcification and thrombosis.

Twelve of the fourteen cases showed thrombi in the larger vessels, i.e., the descending branch of the left coronary or the main trunks. Furthermore, many of the minute branches of the arteries were occluded as a result of atheromatous deposits and this explained the presence of the small fibrous areas seen in many of the hearts.

The microscopic picture of the heart muscle in all of the cases examined (thirteen of the fourteen) was also practically uniform. Sections taken from the site of rupture exhibited necrosis of the muscle fibers with fragmentation and segmentation. Considerable débris was scattered about. The transverse striae of many of the fibers were not visible. Edema was also present in some of the sections. Large numbers of red blood corpuscles were usually present in the interstices of the muscle tissue. In many of the sections an interesting finding was the marked infiltration of leucocytes into the necrotic area. These leucocytes were principally polymorphonuclear neutrophiles with a few lymphocytes. One case (V), in which the rupture occurred at a point where the ventricle wall was quite thick showed scattered foci in which muscle tissue was being replaced by an overgrowth of fibroblasts, associated with the presence of thin-walled blood vessels, i.e., granulation tissue (see Fig. 2).

Adjacent to the area of necrosis there was usually considerable fibrous tissue replacement of the musculature. Evidence of true fatty degeneration of the muscle fibers was present in the zone between the necrotic and fibrotic portions in only five cases; incidentally, all of these five cases showed necrosis and fibrous tissue replacement of the heart muscle at the site of rupture. Practically all of the thirteen cases examined microscopically showed the presence of minute fat droplets scattered about, such as are to be found in many hearts (Masters[34]). In neither of the two hearts (VI and XIII) that grossly presented considerable subepicardial fat and whose musculature appeared somewhat "pale," were there any signs of fatty degeneration or even marked fatty infiltration. Nevertheless, both of these specimens would doubtlessly have been called "fatty hearts" by many of the older investigators and even by some of the present day. Quain, in 1872, noted "fatty degeneration" in seventy-seven of the one hundred cases collected. I lay stress on this point of fatty degeneration in relation to the microscopic findings in the cases here reported, because of the emphasis which was, and still is, placed on "fatty degeneration" as a cause of spontaneous rupture of the heart. Practically the majority of older authors gave "fatty degeneration" as the commonest cause of cardiac rupture. Even today statements may be seen to the effect that "rupture of fatty hearts is a frequent cause of death,"[35] or that "spontaneous rupture of the heart is particularly common in cases of fatty degeneration,"[36] or, again, "In 77 per cent of fatty hearts Hamilton reported spontaneous rupture."[35] It is to be noted, however, that these statements were based on observa-

tions which preceded the introduction of accurate methods for the determination of fatty changes in the heart muscle, such, for example, as that of selective staining.

After examination of the hearts here described, I cannot agree with the above quoted statements, but on the contrary, believe that fatty degeneration and infiltration are the result of the process that produces rupture rather than the cause. In confirmation of this, let me quote Zeigler:[37] "In anemic infarcts of the heart, kidneys and spleen, fatty cells are found in the zone of transition between the necrotic and the living tissue, that is, where the circulation of the blood and lymph is weak, but has not ceased." To substantiate, further, my contention, I quote Wells:[38]

"Fatty degeneration is usually brought about by poisons, while abnormal fatty infiltration depends usually upon decreased oxidation, due to lack of either oxygen or hemoglobin in the blood. If the anemia is extreme, however, the cells degenerate and then we find a true fatty degeneration caused by lack of oxygen. Thus, in an anemic infarct, fat accumulates about the periphery of the dead area, probably because fatty acids and glycerol diffuse in slowly from the surrounding parts where circulation still goes on, and are built up into fat by the cell lipase, for in anemic areas the intracellular oxidases cannot destroy these substances as they normally do, because of lack of oxygen."

Finally, Masters has clearly proved that fat is present in normal human hearts and that what is sometimes diagnosed as "fatty degeneration" is merely the fat normally present.

Naked eye and microscopic examination of the other viscera in all of the fourteen cases failed to show changes bearing upon the question of spontaneous rupture of the heart, although in six of the cases the anatomical lesion of chronic arteriosclerotic interstitial nephritis was found, and in four cases arteriosclerotic changes in the vessels of the brain were observed.

PROGNOSIS AND TREATMENT

Spontaneous rupture of the heart is probably always fatal, no case of repair of such a rupture having ever been reported. Occasionally patients with traumatic rupture of the heart recover following surgical intervention or by spontaneous repair, but this occurs in healthy or normal hearts. It is now generally known that spontaneous rupture occurs only in hearts that are diseased. Hence, it would be useless to attempt to suture the degenerated and necrotic myocardium surrounding the rupture. Some authorities suggest treating the heart tamponade which follows rupture by puncture or aspiration. Most assuredly this would be useless unless plugging of the tear by a thrombus or pericardial adhesions took place, since fresh blood would replace that removed. Should the day come when spontaneous rupture of the heart is diagnosed clinically, of what value will this diagnostic acumen be when one has to stand by helpless and watch the patient die?

The treatment of spontaneous rupture of the heart, whether the lesion be due to the usual cause, i.e., coronary occlusion with its subsequent infarction, or to the unusual, such as ulcerative endocarditis, gumma or cyst, should be the same as suggested by Quain in 1872, namely, "In treatment, the great object should be to secure complete rest and to avoid any active treatment which might excite the heart." The best way to secure rest is by administering large doses of morphine immediately after the diagnosis has been made. I can see no indication for giving digitalis to these patients.

In this day of preventive medicine, it may be in order to suggest that treatment or, better yet, prevention of arteriosclerosis and its sequelae is the best treatment for cardiac rupture.

CASE HISTORIES

Following are brief records of the fourteen cases described in this report, on the examination of which the opinions and conclusions here expressed are founded:

CASE I.—(January 28, 1922, No. 7762.) J. M., a white man, aged eighty-one years, laborer, was admitted to the hospital, complaining of pain in the chest and shoulders. These symptoms were of several weeks' duration and were accompanied by dyspnea, palpitation of the heart, sour eructations, and edema of the legs. The patient had had syphilis and gonorrhea and had always been a heavy drinker.

At the time of admission he was neither dyspneic nor orthopneic. The pupils were equal and regular and reacted to light. The heart was enlarged downward and to the left; the heart sounds were distant but regular; no murmurs were audible. The radial arteries were easily palpated. Blood pressure 140/90 mm. Urinalysis—specific gravity 1.024; albumin trace; glucose positive; granular casts in moderate numbers. The temperature was normal.

The day after admission the patient's temperature was 102° F., pulse 80, respirations 22. Dullness was elicited over the right lower lobe with tubular breathing and increased vocal resonance. Many coarse and fine râles were heard over the same area.

The following day the temperature was normal. On the third day after admission, while at the noonday meal, the patient became asphyxiated on a morsel of food. Tracheotomy was performed, but the patient died.

At necropsy, performed by Dr. Morton Ryder, the pericardial sac contained about 300 c.c. of partially clotted blood lying anterior to the heart and obscuring it from view. The epicardial surface was smooth. The heart was enlarged, weighing 560 grams. Two centimeters above the apex, on the anterior surface of the left ventricle, was a laceration, 8 mm. in length, the edges of which were discolored by blood. The heart muscle in this region was yellowish brown in color, friable, and in a few places infiltrated by blood. A probe inserted through the tear passed obliquely into the cavity of the left ventricle. A small grayish red thrombus was attached to the inner wall of the ventricle at this point. The apical portion of the left ventricle was the site of an anemic infarction; the muscle of the remaining upper portion was moderately hypertrophied, reddish brown in color, and of average firmness. The valves of the heart were normal except for slight thickening of the aortic cusps near their bases and of the aortic leaflet of the mitral valve. In the left coronary artery, about 4 cm. from its origin, was a pale grayish red thrombus occluding the lumen, firmly attached to the vessel wall and extending over a

distance of 1 cm. Below this point the vessel was intact. Both coronary arteries were somewhat constricted at their orifices and were studded throughout their extent with raised yellow plaques, some of which were calcified. The descending aorta presented marked atherosclerosis with calcification.

The brain showed atrophy and pial edema, and the cerebral arteries were sclerosed. The kidneys showed the picture of interstitial nephritis.

Microscopic Examination.—(Hematoxylin-eosin.) Section of the heart muscle, taken from the point of rupture, showed extensive infiltration by red blood cells, the infiltrated areas being limited externally by necrotic muscle fibers, between which were quantities of nuclear débris.

(Sudan III.) Section taken from the same point showed the presence of minute fat droplets in small groups of muscle fibers lying in proximity to the areas of necrosis. The majority of the degenerated muscle fibers, however, were free from fat.

The left coronary artery exhibited marked sclerosis and calcification, with thrombosis and almost complete occlusion of the lumen.

Section of the aorta (ascending part) showed a normal vessel wall.

CASE II.—(January 30, 1922, No. 7770.) M. W., aged sixty-six years, single, was found dead in bed in a rooming house. No further history was obtainable. There were no external signs of violence.

At autopsy, the pericardial sac was distended with 400 c.c. of clotted blood, which was easily removed, leaving some yellowish gray fibrous strands adherent to the anterior surface of the heart. The heart was enlarged, weighing 450 grams. There was a tear 3 cm. in length on the anterior wall of the left ventricle near the apex. The ventricular wall was 7 mm. thick at the site of rupture and was infiltrated by blood. About 3 cm. below the origin of the ramus descendens of the left coronary artery was a brownish red thrombus, 4 mm. in length, occluding the lumen of the vessel. This vessel, together with the circumflex branch, presented numerous yellow plaques and calcification of the arterial wall, with diminution of the caliber of the lumen. There was some thickening of the bases of the aortic cusps and of the medial mitral curtain. The descending aorta presented a number of scattered yellow plaques and, near its bifurcation, a few areas of calcification and ulceration. In the upper abdominal portion there were a few grayish plaques with longitudinal striae.

The kidneys presented the picture of chronic interstitial nephritis. The prostate was uniformly enlarged. The testicles showed bilateral interstitial scar tissue formation which, on microscopic examination, revealed the changes incident to chronic interstitial syphilitic orchitis.

Microscopic Examination.—Section of the heart muscle showed areas of fibrosis with many small, thickened and partially occluded blood vessels in the same areas. Section of the ascending portion of the aorta presented the picture of syphilitic aortitis.

CASE III.—(February 17, 1922, No. 7813.) This case presents a most unusual and interesting history. It was reported by Dr. Paul Reznikoff under the title of "The Auscultatory Sign in a Case of Spontaneously Rupturing Heart." The interest of his article makes it worth quoting verbatim:

"A white man, aged forty-three, American, married, an embosser, admitted to Bellevue Hospital February 16, 1922, stated that he had always been in perfect health, never having had a sick day, until February 6th. On that day he had a constricting pain over the lower part of his left chest, which was diagnosed as indigestion. Following this he had six such attacks in the next ten days, each succeeding one more severe and lasting longer. In the last few the pain radiated to the left supraclavicular region and down the ulnar margin of the left forearm.

These spells of sharp pain occurred whether he was active or in bed, and were not relieved by any of the many remedies tried. The attack for which he came into the hospital was his seventh and had begun the night before, never remitting.

"The patient was rather well developed and of fair nutrition. When he reached the ward he was moderately cyanosed, orthopneic and in marked distress. He groaned constantly and could not lie quietly on his bed. Neither the nitrites nor morphine were of any avail in allaying his terrific discomfort. The heart was slightly enlarged to the left and right, the apex beat being felt in the fifth left intercostal space, 11 cm. from the midsternal line. No thrills were present and the apex impulse was localized and of fair strength. The heart sounds were of poor quality. The rate was slow, averaging 72 a minute, and there were no murmurs present. At the base he had an accentuated aortic second sound. The rhythm was peculiarly irregular. About eight beats would occur at a definite, regular rate. This series would be followed quite abruptly by about eight beats at a different rate, but forming a regular unit by themselves. An electrocardiographic record substantiated this finding. It was interpreted by Dr. Cary Eggleston as either a phenomenon of two foci for stimuli, i.e., a normal sinus and a focus with a slower rate near the sinus, or as an alternating bradycardia and tachycardia from the same focus. (See Fig. 3.) The patient's arteries were thickened and tortuous wherever they could be examined. The blood pressure was systolic, 170; diastolic, 120. No other physical findings had any bearing on the cardiovascular condition.

"The course of the disease was short and stormy. The pain was unrelenting. The patient was becoming more dyspneic. A cold perspiration bathed him continually and he was a picture of utter misery. At 7 P.M., while under observation, the unfortunate man threw his head back, and his face expressed the most extreme anguish. He had a sudden attack of projectile vomiting, became markedly cyanosed and collapsed. His wrist pulse was imperceptible. He took four deep inspirations about thirty seconds apart and then died.

"During these two minutes a stethoscope was clapped to his chest and over the precordium was heard an extraordinary sound. A continuous, muffled, low-pitched, rushing rumble, louder during the expiratory phase, dimmed during the few inspiratory periods, was constantly heard. No heart sounds were evident. All this transpired so quickly that only two auditors could listen to this remarkable murmur. Its significance was not apparent, and at first it was imagined by the observers that the sounds were such as might be heard over a fibrillating ventricle.

"Necropsy, performed by Dr. Douglas Symmers, revealed an irregular laceration of the wall of the left ventricle in immediate proximity to the interventricular septum, situated midway between the base and apex of the heart [see Fig. 6]. This laceration was 3 cm. in length and ran obliquely downward and toward the right directly over the course of the corresponding coronary artery. The fat tissue covering the upper and outer half of the conus arteriosus was infiltrated by blood, the infiltrated area measuring 5 cm. in length and 3.5 cm. in breadth. From the extreme upper end, a broad linear area of hemorrhage extended upward toward the opening of the corresponding coronary artery. The left coronary artery was opened by the pathologist for a short distance and presented at a point 1.5 cm. from its origin a calcareous plaque. The laceration extended through the heart muscle and opened into the ventricle. The muscle tissue in the immediate vicinity of the lacerated area was soft, almost fluctuating, for a distance of about 1 cm., and this area of softening extended downward in the general direction of the apex for a distance of 2.5 cm. A transverse section of the heart muscle was made just above the area of laceration. Here the coronary artery was about 2 mm. in diameter and markedly calcified. The lumen could be seen filled with a reddish body, about the size of a horsehair, probably a fresh thrombosis. The pericardium was filled with fluid and freshly clotted blood.

"In the light of the necropsy, the clinical course was thus interpreted: The acute onset of illness, the continuous, agonizing precordial pain and distress all point to the rather sudden thrombosis of the calcified coronary and the resulting degeneration of the heart muscle. The sudden collapse, projectile vomiting and immediate death were probably coincident with the rupture of the heart. There can be little doubt that the auscultatory evidence of this dramatic accident was the rumble heard as the blood poured into the pericardium, so like the sound of water rushing through a bursting dam."

The gross pathological findings in the other organs were as follows: Hemochromatosis of the liver and pancreas; marked congestion of the kidneys and spleen; brown induration of the lungs.

Microscopic Examination.—(Hematoxylin-eosin.) Section of the heart muscle from the point of rupture exhibited marked hemorrhagic infiltration; adjacent to this area there was acute necrosis of the muscle fibers, with slight leucocytic infiltration. Section of the ramus descendens of the left coronary at the site of rupture showed marked calcification of the vessel wall, with incomplete occlusion of the lumen by an organized and canalized thrombus.

(Sudan III.) No evidence of fat was found in any of the sections.

The aorta showed slight sclerosis of its intimal layer.

CASE IV.—(Spring of 1922.) An obese white man, about fifty years of age, laborer, was admitted to Bellevue Hospital in the Spring of 1922, complaining of cough and dyspnea. He was up and about the ward for several days, apparently quite comfortable, until the morning of his death, when he suddenly became dyspneic, cyanotic and complained of excruciating precordial pain. He was put to bed immediately and died a few hours later.*

At necropsy, the heart was enlarged, weighing 520 grams. There was bulging and thinning of the posterior surface of the left ventricular wall near the apex. An irregularly stellate perforation, the longest radiation of which measured 2.5 cm., was present at the thinnest portion of the ventricle wall. The aneurysmal cavity was filled with a firmly adherent blood clot (see Fig. 7). The remainder of the left ventricle was hypertrophied. The visceral layer of the pericardium covering the area of rupture was irregularly coated with dried blood and fibrin. About 5 cm. from the origin of the left coronary artery, the vessel was almost completely occluded by a calcified plaque. The right coronary vessel was likewise markedly sclerosed. The aorta showed many irregular yellow plaques. Near the origin of the descending aorta an aneurysmal dilatation of the vessel wall was visible.

Microscopic Examination.—(Hematoxylin-eosin.) The heart muscle from the aneurysmal portion of the left ventricle showed few muscle fibers but enormous quantities of free blood and extensive fibrous replacements.

(Sudan III.) In the same section, numerous heart muscle fibers in the immediate vicinity of areas of necrosis were packed with minute fatty particles. The muscle tissue in remote parts was entirely free from fat.

The left coronary artery was fibrotic, the lumen being reduced to a mere slit. The adventitia was infiltrated by red cells. Section of the aorta exhibited moderate sclerosis with atheromatous changes in the subintimal layers.

CASE V.—(September 27, 1922.) F. K., a white man, fifty-five years old, was found dead lying across his bed. No other facts were obtainable.

Necropsy, performed by Dr. Charles Norris, showed the pericardial sac filled with fluid blood. The heart was slightly enlarged, weighing 380 grams. There was a transverse tear, 3 cm. long, on the anterior surface of the left ventricle, about

*It is impossible to find any record of this case in the hospital record room. The specimen described below was identified by Dr. Harry A. O'Connor, who performed the autopsy, and to whom I am also indebted for the information given above.

2.5 cm. above the apex, with a small tortuous perforation leading into the ventricular chamber (see Fig. 2). The left ventricle presented hypertrophy of its wall throughout practically its entire extent. The coronary arteries were sclerosed and calcified. The anterior coronary artery was occupied by a thrombus which occluded the vessel for a considerable distance. The aorta showed several large smooth plaques and the aortic cusps were sclerosed and calcified.

The kidneys presented the picture of chronic interstitial nephritis.

Microscopic Examination.—(Hematoxylin-eosin.) The heart muscle showed scattered foci in which muscle tissue was being replaced by overgrowth of fibroblasts, associated with the presence of a number of thin walled blood vessels, in other words, granulation tissue. In other places the muscle fibers were replaced by fibrous tissue.

The lumen of the anterior coronary artery was almost completely occluded by a reticulated thrombus made up of hyaline material, in the interstices of which were variable numbers of red cells. The media was extensively replaced by hyaline connective tissue in which were large and small necrotic foci containing cholesterin crystals. The adventitia was thickened, in places infiltrated by lymphocytes, in other places fibrotic or calcified.

(Sudan III.) No evidence of fatty degeneration visible.

A section of the aorta presented marked sclerosis and calcification, involving both intima and media.

CASE VI.—(October 8, 1922.) J. G., an obese white woman, sixty years of age, was suddenly taken ill four days prior to her death, complaining of headache, vomiting and severe "heart pain." Autopsy was requested by her son after the family physician had given the cause of death as "ptomaine poisoning complicated by chronic endocarditis."

At necropsy, which was performed by Dr. Charles Norris, the pericardial sac was found to be occupied by a blood clot weighing 300 grams. The heart was slightly enlarged, weighing 370 grams, soft and flabby; epicardial fat was abundant. There was a tear 4 cm. long on the posterior surface of the left ventricle, adjacent to and parallel with the interventricular septum and posterior coronary artery. For a distance of 1 cm. to the left of the laceration, the heart wall was denuded of pericardium, exposing a ragged area of heart muscle which resembled a thrombus (see Fig. 5). The wall at this site was thin, measuring 2 to 3 mm. The right coronary artery ran just to the right of the tear; the vessel was readily palpated because of sclerosis of the vessel wall. A hemorrhagic area, 1 cm. in diameter and 1.5 cm. in depth, was present about 2 cm. above and to the right of the rupture and involved the interventricular septum. Three centimeters from its origin, the right coronary artery was thrombosed, and there was almost complete obliteration of its lumen. The orifices of both coronaries were constricted by yellowish plaques. The left ventricle was slightly hypertrophied, the musculature pale and flabby. The papillary muscles were tabby cat in appearance. The arch of the aorta was apparently normal, but the thoracic and abdominal portions were markedly calcified, the intima showing several red thrombi.

Microscopic Examination.—(Hematoxylin-eosin.) Section of the heart muscle from the point of rupture showed extensive hemorrhagic infiltration with destruction and replacement of muscle fibers by fibrous tissue.

(Sudan III.) No evidence of fatty degeneration.

The right coronary artery was calcified and hyalinized and at one point was almost completely occluded by a mixed thrombus, apparently of recent origin.

The abdominal aorta showed extensive replacement of the media and intima by sclerotic and hyalinized connective tissue.

CASE VII.—(October 10, 1922.) J. B., white, male, about sixty years of age, was found dead on the floor of a room in a boarding house. On a table in the same room stood an empty whiskey bottle. No further history was obtainable.

Autopsy was performed by Dr. Charles Norris. No external signs of injury were present. The pericardium was filled with clotted blood, weighing 240 grams. The heart was enlarged and weighed 440 grams. On the anterolateral aspect of the left ventricle, 5 cm. from the apex of the heart, was a tear, 1 cm. in length and apparently incomplete, it being possible to insert a probe for a distance of only 5 mm. On cross section, however, the laceration was seen to communicate with the left ventricle after a somewhat tortuous course. Immediately adjacent to the tear the muscle was infiltrated with blood, but lateral to this area the muscle was considerably lighter in color than the rest of the ventricle and friable. The left ventricle was hypertrophied and dilated, and areas of fibrosis were visible throughout the heart wall. There was thinning of the left ventricular wall close to the interventricular septum, the result of an old infarct, the average width at this site being 8 mm. The circumflex branch of the left coronary artery was calcified and occluded at a point 9 cm. from its orifice. This was the vessel of supply to the area of rupture. The descendens branch of the left coronary artery was occluded by an old thrombus 3.5 cm. from its origin, the thrombosed area being 2 cm. in length, causing bulging of the vessel at this point, returning to normal caliber further down its course (see Fig. 4). This vessel supplied the area of old infarction described above. The aorta presented innumerable raised yellowish plaques throughout its entire length.

Microscopic Examination.—(Hematoxylin-eosin.) There was extensive replacement of muscle fibers by fibrous tissue in a section taken near the old infarct. Necrosis with leucocytic infiltration was visible at the point of rupture.

(Sudan III.) No evidence of fatty degeneration.

There was complete obliteration of the lumen of the circumflex branch of the left coronary by sclerosis and hyalinization of the vessel, with scattered plaques of calcification. The descending branch of the left coronary presented an old thrombus which occluded the markedly constricted lumen of the vessel, sclerosis and calcification of the vessel wall being prominent.

Section of the aorta showed the histological changes of syphilitic aortitis.

CASE VIII.—(January 7, 1923.) The subject was a white man about sixty-five years of age, dressed in workman's clothes, who was found dead in a comfort station, having apparently fallen off a toilet seat at the time of death. No marks or bruises were visible on the body.

At autopsy, which was performed by Dr. Charles Norris, the precordial area was enlarged, the pericardial cavity containing a large amount of clotted blood. The heart was apparently normal in size and weighed 340 grams. A tear, 2.5 cm. long, running vertically, was visible several centimeters above the apex, extending through the anterior wall of the left ventricle. A small thrombus was adherent to the inner wall of the heart at the site of rupture. The orifices of the coronary arteries were apparently normal, but areas of calcareous thickening were scattered throughout their course, especially the ramus descendens of the anterior coronary, which was almost completely obliterated by a calcareous plaque located 5 cm. from its orifice. There was no apparent hypertrophy or dilatation of any of the chambers. All the valves were slightly sclerosed; the right anterior and posterior cusps of the aortic valve were fused. The aorta presented a few calcareous plaques on its intimal surface, but the vessel wall was still elastic. The lower portion of the abdominal aorta showed an atheromatous patch about 5 cm. in diameter.

The brain revealed a few plaques of sclerosis in the middle cerebral vessels. The kidneys were apparently normal.

Microscopic Examination.—(Hematoxylin-eosin.) Section of the heart wall, taken near the site of rupture, exhibited hemorrhagic and leucocytic infiltration of the muscle tissue with areas of necrosis.

(Sudan III.) A slight degree of fatty degeneration was present in the immediate vicinity of the necrotic area.

The left coronary artery showed sclerosis with calcification of the media and narrowing of the lumen of the vessel.

Section of the arch of the aorta showed a well preserved vessel wall.

CASE IX.—(March 12, 1923, No. 8662.) A. E., a woman fifty years of age, single, white, was admitted to the hospital on February 20, 1923, complaining of weakness of the right arm and leg and of the right side of the face. Her mother died of apoplexy and one sister of heart failure. The patient, who had been a school-teacher until two years previously, stated that she had not been well for the past two years, complaining of headaches, blurred vision and attacks of dizziness, palpitation of the heart and occasional pain over the heart. Menopause at forty-five years. The patient had been told that her blood pressure was abnormally high, and this caused her to discontinue her work as a teacher. The night before admission to the hospital, she fell to the floor and was not able to arise; she was not unconscious.

Physical examination at the time of admission to the hospital revealed a plethoric, obese woman, with upper and lower right extremities powerless and spastic. Right facial palsy was complete. All the reflexes were exaggerated; those on the right side more so than on the left; Babinski's sign was present on the right. Ophthalmoscopic examination showed arteriosclerosis of the retinal vessels. The heart was slightly enlarged to the left. The heart sounds were muffled and distant; the rhythm was regular; no murmurs were heard. The pulse was hypertensive in character, the radial arteries slightly thickened. Blood pressure—190/110. The urine was normal; Wassermann negative.

On the morning of March 11, the patient became incontinent, but otherwise felt quite comfortable. At 4:30 A.M. on March 12, she was found dead in bed. She had made no complaint all night, having had a bowel movement at 2 A.M. The preautopsy diagnosis was coronary thrombosis or embolism.

At necropsy, performed by Dr. Steele, the pericardial sac contained 200 c.c. of blood. On the anterior surface of the heart, midway between the apex and the auriculoventricular junction, was a horizontal rent, 15 mm. long, communicating with the cavity of the left ventricle. At the point of rupture, the wall of the left ventricle was thin, forming an aneurysmal dilatation about 2.5 cm. deep and wide. A firmly attached thrombus partially filled this pouch. The heart muscle immediately adjacent was infiltrated by blood. The heart weighed 320 grams. The muscle was poor in quality, showing fibrous streaks and patches. On opening the heart, the endocardium and valves on the right side were normal. The mitral valve was sclerosed. The aortic cusps were sclerosed at their bases and the wall of the left ventricle adjacent presented a few atheromatous areas. The descending branch of the left coronary was occluded near its origin by calcific encroachment on the lumen.

The kidneys presented the picture of chronic interstitial nephritis. There was softening of the brain in the region of the left lenticular nucleus and the posterior limb of the internal capsule. Atherosclerosis of the abdominal aorta and common iliacs, vertebrals and basilar artery was marked.

Microscopic Examination.—(Hematoxylin-eosin.) At the point of rupture the heart muscle showed signs of necrosis and the remaining fibers were separated by hemorrhagic and leucocytic infiltration. Sections taken several centimeters from the rupture showed considerable fibrous replacement of the muscle tissue.

(Sudan III.) There was evidence of marked fatty degeneration of the muscle fibers in the immediate vicinity of the necrotic area.

There was marked sclerosis of the left coronary vessel, with almost complete obliteration of the lumen by an area of calcification. Section of the aorta presented considerable sclerosis of the subintimal tissues.

CASE X.—(April 21, 1923.) A. P., seventy-two years old, garage worker, was found dead on April 20, 1923, at his place of employment. He had just finished working and was on his way to the washroom when he dropped dead.

Autopsy was performed by Dr. Charles Norris. There were no external signs of injury, although there was a fracture of the mandible with laceration of the buccal mucous membrane, due to the fall on the concrete floor at the time of death. The pericardial sac contained 250 grams of blood. The arch of the aorta was dilated. The heart was hypertrophied and weighed 420 grams. On the posterior surface of the left ventricle, 4 cm. from the interventricular sulcus, was a tear 1 cm. in length, which appeared to be superficial but, upon passing a dull probe into it, the instrument emerged into the cavity of the ventricle just posterior to the papillary muscle. On cross section, the rupture was seen to be complete, the wound internally being about 4 mm. in length. The muscle adjacent to the rupture was infiltrated by blood. The left ventricle was dilated, the wall being much thinner than in normal circumstances. The valves were apparently normal. On the arch of the aorta were a few yellow plaques; the thoracic and abdominal portions showed moderate sclerosis. The coronaries were thickened and tortuous; the posterior branch of the right coronary showed scattered plaques of sclerosis and its lumen was considerably diminished in caliber. The orifices of both right and left coronaries were not encroached upon.

The cerebral blood vessels were slightly sclerosed. The kidneys presented the picture of chronic diffuse nephritis.

Microscopic Examination.—(Hematoxylin-eosin.) There was extensive fibrous replacement with necrosis and leucocytic infiltration of the ventricular wall adjacent to the rupture.

(Sudan III.) No evidence of fatty degeneration was present in the above section.

The right coronary artery showed sclerosis and beginning calcification, with encroachment on its lumen.

The aorta presented extensive sclerosis and calcification with areas of necrosis, particularly in the subintima.

CASE XI.—(November 30, 1923, No. 9376.) M. D., aged fifty-six, laborer, was admitted to the hospital on November 23, 1923, complaining of shortness of breath of two weeks' duration. The onset was gradual, but the symptoms became worse during the three days prior to admission. The patient described his sensations as a feeling of suffocation, accompanied by dizziness and a cough productive of brownish red sputum. No other complaints were elicited. The patient gave a history of having been a moderate drinker of whiskey. As far as he could remember, he had never been ill before. He stated that he had had syphilis and gonorrhea thirty years previously. His father died at seventy years of age from "fatty degeneration of the heart"; his mother died of "old age" at eighty-six years.

Physical examination showed a fairly well developed elderly looking white man who was dyspneic and orthopneic, but not cyanotic. The pupils were equal and regular and reacted to light and accommodation. The chest was of the emphysematous type. There was slight dullness at the bases of both lungs, coarse moist râles and diminished breath sounds. The apex beat of the heart was palpable in the sixth space, one-half inch to the left of the nipple line; no thrills were

palpable. There was a blowing systolic murmur over the mitral area. The heart's rhythm was regular, the rate rapid (108) and the sounds of poor quality. The radial, brachial and temporal arteries were palpable. Abdominal examination elicited an enlarged liver, the lower border being at the level of the umbilicus; no pulsation was felt nor tenderness elicited. Examination of the urine showed a faint trace of albumin and a few hyaline casts.

The day after admission a gallop rhythm was heard at the apex of the heart, but the patient was fairly comfortable and had no complaints. However, at 9:15 P.M. he suffered a severe attack of dyspnea and died.

Necropsy was performed by Dr. Stanley-Brown. The pericardial sac was filled with clotted blood. The heart was markedly enlarged, weighing 700 grams. On the anterior surface of the left ventricle, near the apex, there was a distinct dilatation of the heart wall, in the center of which a ragged tear 3 cm. long was visible. This laceration communicated with the left ventricular chamber. On the inner surface of the ventricle and corresponding to the site of the rupture, was a large thrombus, firmly adherent to the ventricular wall. The pericardial surface adjacent to the tear was infiltrated with blood and presented a shaggy appearance. The muscle wall of the apical portion of the heart was considerably diminished in thickness and friable. Numerous patches of scar tissue were visible in the wall of the upper third of the left ventricle. There was a large area of scar tissue in the left ventricular chamber adjacent to the interventricular septum, the result of an old infarction. The wall at this site was only a few millimeters in thickness and was completely fibrotic. The valves were normal in appearance. The coronary arteries were markedly sclerosed on palpation. The descending branch of the left coronary artery was completely occluded about 5 cm. from its mouth by an organized thrombus of grayish red color. The circumflex branch supplying the fibrous area described above was also completely occluded, the lumen of the vessel being obliterated by sclerosis and calcification of the wall of the artery itself. The arch of the aorta was markedly sclerosed.

The liver was markedly congested. The kidneys were normal in size, but the capsules stripped with difficulty, leaving rough, granular surfaces.

Microscopic Examination.—(Hematoxylin-eosin.) Section of the heart muscle taken from near the point of rupture revealed marked necrosis of the muscle fibers with hemorrhagic and leucocytic infiltration. Section taken from the area of old infarction showed marked fibrous replacement.

(Sudan III.) There was moderate fatty degeneration of muscle fibers in the immediate neighborhood of the fibrotic areas, the fatty areas being scattered.

The ramus descendens of the left coronary artery showed extensive sclerosis and hyalinization of the vessel wall with complete occlusion of the lumen by a recent red thrombus. The circumflex branch was also markedly sclerosed and its lumen was obliterated by an old hyaline thrombus.

The ascending aorta presented sclerosis and calcification of the subintimal and muscle layers.

CASE XII.—(February 5, 1924, No. 9568). D. C., white, male, eighty years of age, was found dead at his place of residence. Fully dressed, he lay face downward on the floor of his bedroom. No further history was obtainable.

Autopsy was performed by Dr. Weeks. There was a contusion over the bridge of the nose and another above the right eye, probably the result of falling on his face at the time of death. The pericardial sac contained about 250 c.c. of fluid and clotted blood. The heart was enlarged, weighing 450 grams. On the anterior surface of the left ventricle, 5 cm. from the apex, was a tear measuring 16 mm. in length. Internally the rupture was ragged and of greater length than externally; one of the columnae carneae was involved in the rupture. The myocardium presented

small scattered areas of fibrosis and was infiltrated with blood and friable at the point of rupture. The coronary arteries were irregularly sclerosed, but no thrombus could be located in the main vessels or their branches. The abdominal and thoracic portions of the aorta showed marked sclerotic changes and beginning aneurysmal dilatation of the arch of the aorta.

Microscopic Examination.—(Hematoxylin-eosin.) Section of heart muscle taken from an area adjacent to the rupture, showed extensive necrosis of the muscle fibers, with hemorrhagic and leucocytic infiltration. Section of the heart muscle from the upper portion of the left ventricle presented marked fibrous replacement.

(Sudan III.) Evidences of very slight fatty degeneration were present in the muscle tissue adjacent to the necrotic area.

Both the right and left coronary arteries were extensively sclerosed and hyalinized, with marked diminution in caliber of the lumina of the vessels.

Section of the ascending aorta showed extensive sclerosis of the intima and media.

CASE XIII.—(March 21, 1924, No. 9664.) M. S., sixty-five years old, male, was found dead on the fourth floor of a tenement house, about one yard from the top of a stairway, which he had apparently ascended just prior to his death. A cousin who found him there had seen him about three months previously, at which time the patient had apparently been in good health. Two weeks prior to death, he had written to this same relative, stating that he had been sick in bed for three days, but did not state what had ailed him or any of his symptoms. No further history was obtainable.

Autopsy was performed by Dr. Kaplan. No external signs of violence were found. On opening the pericardial sac, about 250 c.c. of fluid blood escaped, leaving a large clot weighing 350 grams in the dependent portion of the sac. The heart was markedly enlarged, weighing 500 grams, and was covered by a thick layer of fat. In the apical region was an irregular, ragged tear 7 cm. in length, extending to both the anterior and posterior walls of the left ventricle (see Fig. 1). Surrounding the rupture was an area of muscle tissue which was thin and friable and which formed a distinct aneurysmal dilatation of the apex of the left ventricle. The two pericardial layers were adherent for a distance of several centimeters lateral to the tear, but were easily separated, leaving shaggy surfaces. The left ventricle was markedly hypertrophied; the musculature was pale yellowish brown in color, especially the papillary muscles of the left ventricle. The coronary vessels were sclerosed; the descending branch of the left coronary artery was completely occluded 5 cm. from its origin by a grayish red thrombus. The aorta presented numerous fatty and sclerotic plaques throughout its course.

There were no noteworthy changes in any of the other organs except marked congestion and edema of the lungs.

Microscopical Examination.—(Hematoxylin-eosin.) Section of the heart muscle from a point near the rupture showed extensive necrosis of the muscle fibers, with hemorrhagic infiltration. A section taken 2.5 cm. from this point revealed fibrous replacement.

(Sudan III.) No evidence of fatty degeneration was visible.

The ramus descendens of the left coronary artery presented marked fibrosis of the vessel wall, with obliteration of the lumen by a recent thrombus.

Numerous calcific and sclerotic plaques were present in the aorta.

CASE XIV.—(August 11, 1924.) J. M., white, male, sixty-eight years of age, no occupation, was found dead seated on a toilet and leaning over the edge of a bath tub. He was fully dressed when found. His housekeeper said that he had been drinking heavily for some time prior to his death; otherwise no history was obtainable.

At autopsy, performed by Dr. Gonzales, the body was markedly decomposed. Cyanosis of the face and head was prominent. There was a large amount of blood in the pericardial sac which upon removal, left a decomposed heart of about normal size. On the anterior wall of the left ventricle, about midway between apex and base, was an irregular tear, about 2 cm. in length, that communicated directly with the ventricular chamber. The wall adjacent to the rupture was infiltrated by blood and was softer than the rest of the heart. The endocardial aspect of the laceration was covered by a thrombus. The left coronary artery was occluded near its origin by a thrombus. Both coronary vessels were markedly sclerosed throughout. The aortic cusps were calcified and a small aneurysm was present at the arch of the posterior coronary artery. All the valves of the heart were sclerotic. The aorta showed marked sclerosis.

No microscopic sections of the heart were made, in view of the marked decomposition of the organ.

SUMMARY

1. Fourteen cases of spontaneous rupture of the heart are analyzed and described.

2. The condition is not a rare one but is frequently overlooked.

3. Eleven (78 per cent) of the patients were representatives of the laboring class; no members of the professional class were represented.

4. The male sex predominated (86 per cent), twelve of the fourteen subjects being males.

5. The average age was sixty-three years. The youngest individual was forty-three years old, the oldest eighty-one.

6. Syphilis was not an etiological factor in any of the cases of this series.

7. Arteriosclerosis was the predominant factor in all of the fourteen cases.

8. A thrombus was found in twelve of the cases (86 per cent); the ramus descendens of the left coronary artery was thrombosed in nine instances, the main trunk in one, the circumflex branch in one, and the right coronary in one.

9. Nine (65 per cent) of the hearts ruptured while in the stage of acute infarction.

10. Microscopically the site of rupture exhibited necrosis of the muscle with infiltration of red blood cells and leucocytes.

11. True fatty degeneration was present in five of the fourteen cases, representing the sequel to infarction and necrosis rather than the cause of rupture.

12. Six of the cases (43 per cent) presented arteriosclerotic interstitial nephritis; four (28 per cent) showed arteriosclerotic changes in the vessels of the brain; otherwise the findings in the viscera were irrelevant.

13. Spontaneous rupture of the heart is always fatal, but death is not necessarily instantaneous.

14. Complete rest, induced by giving large doses of morphine, is the treatment indicated.

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THE OCCURRENCE OF AURICULAR BEATS DUE TO STIMULATION OF THE AURICLES BY THE CONTRACTING VENTRICLES DURING COMPLETE HEART-BLOCK*

A Case Report

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THE following case of heart block is reported because of the occurrence of auricular contractions due to stimuli received from the contracting ventricles:

Mr. C. B., a letter carrier, aged fifty-one years, was observed from Dec. 24, 1924, to April 10, 1925. He complained of substernal pain and breathlessness on exertion. The previous history is irrelevant, except for rheumatic fever at 15, tonsillitis at 39, and chronic otitis media for 30 years. Venereal infection was denied. On Nov. 7, 1924, while carrying letters and feeling well, he was suddenly seized with severe pain beneath the lower sternum. The pain was constant at first, did not radiate and was made worse by exertion. After a week's rest the pain was present only on exertion. There was breathlessness also on exertion. A slow pulse was noted soon after the onset of symptoms.

Examination revealed good nutrition, moderate pulmonary emphysema and a moderate degree of arteriosclerosis. Dyspnea, cyanosis and edema were absent. The cardiac dullness extended 11.5 cm. to the left of the midline in the fifth interspace. Over the entire precordium was heard a blowing systolic murmur, loudest at the apex and transmitted into the axilla. The ventricular rhythm was regular; the rate was 34 to 42 per minute, and was not influenced by exercise. The systolic blood pressure was 170 mm., the diastolic 65 mm. Hg. There were no signs of congestion of the lungs or liver. The urine had a high specific gravity and contained albumin and casts. The Wassermann reaction of the blood was negative.

Frequent electrocardiograms were made. The record of Dec. 24 showed auriculoventricular heart block of varying degree, 2:1, 3:1 and complete. On Dec. 26 there was complete block, with an occasional response of the ventricles to the auricles. The curves of Dec. 27, 30, and 31 and of Jan. 2 and 3 showed complete block. On Jan. 15 there was 3:1 block, with the following response to 1/50 grain of atropine sulphate intravenously:—

	AUR. RATE	VENT. RATE	BLOCK
Before	96	32	3:1
4 min. after	116	41	Complete
7 " "	105	35	3:1
15 " "	105	35	3:1
30 " "	96	36	Complete

On Jan. 24 there was 2:1 block with a period of 3:1 block. On Feb. 16 there was complete block with a short period of 2:1 block. The curve of April 10 showed

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normal A-V sequence with a P-R interval of 0.20 sec., a rate of 85 and defective intraventricular conduction. At this time the patient reported that he had been working and riding a bicycle for several weeks, and had been free from symptoms.

It was thought that the patient had suffered a sudden occlusion of one of the branches of the coronary artery, of arteriosclerotic origin, the infarct involving the His bundle and causing heart-block.

While the block was complete, there was considerable variation in the form of the ventricular complexes, which were usually abnormally broad (an example of this is seen in Lead II of Fig. 1); while the A-V block was partial or absent the ventricular complexes were fairly constant in form; they were abnormally broad and indicated defective intraventricular conduction. According to Wilson and Herrmann,[1] "complete block associated with ventricular complexes of varying form is usually due to bilateral bundle-branch block and not to a lesion of the main stem of the His bundle."

The electrocardiogram of Dec. 30 is reproduced in Fig. 1. The five parts of Lead II are directly continuous. The record shows complete A-V block in all leads. The ventricular complexes are abnormally broad. Throughout the record the auricular cycles during which ventricular contractions occur are of somewhat shorter duration than those during which ventricular beats do not occur. An exception to this appears when the ventricular impulse temporarily arises in a new center, giving rise to the waves marked R′. These R′-waves are followed by an acceleration of the original ventricular pacemaker and a slowing of the auricles. The variation in the intervals between auricular beats makes it impossible to estimate accurately the prematurity of certain abnormal auricular beats.

The eighth, tenth and twenty-second auricular waves of Lead II are inverted and somewhat premature. The second, fourth and sixth auricular waves of Lead III are also premature and inverted. These deflections are marked P′. Each of these premature and inverted auricular waves bears a definite relationship to the preceding ventricular wave, following it by 0.24 sec., with one exception in which the R-P′ interval is 0.23 sec. These waves are premature, but by a rather short interval; they occur late in auricular diastole. They are obviously not ordinary auricular extrasystoles. They are, therefore, regarded as due to stimulation of the auricles by the contracting ventricles. According to Lewis[2] inverted auricular deflections of this form indicate that the auricular beats arise in the lowest level of the auricular musculature, that is, in or near the A-V node.

The sixth and nineteenth auricular deflections of Lead II are abnormal in that they are definitely flatter than the normal auricular waves. They are marked X. They follow the preceding ventricular deflections by intervals of 0.22 and 0.21 sec., respectively. They may be premature by a very small interval. They are intermediate in form between the

normal upright P-waves and the abnormal inverted P′-waves. Lewis[2] has shown that auricular waves of this form result from impulses arising in what he terms the central zone of the auricles. Lewis and White[3] have shown that auricular deflections transitional in form re-

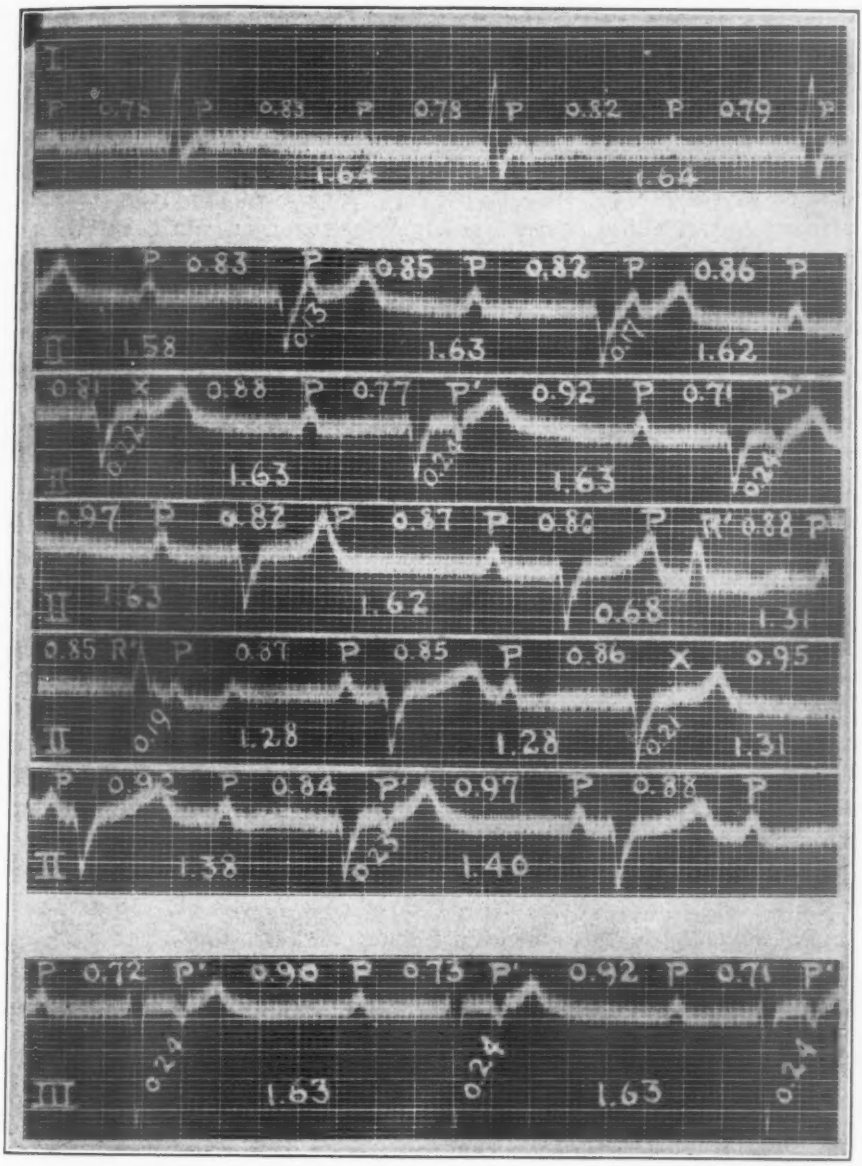

Fig. 1.—Electrocardiogram of Dec. 30, 1924. The interauricular intervals appear above the curves, the interventricular below. The intervals between some of the ventricular complexes and the following auricular complexes are inserted below the curves. The five parts of Lead II are directly continuous. The curve shows complete A-V heart-block. Lead II contains three inverted and premature auricular waves (*P′*) and two transitional auricular waves (*X*) and two ventricular complexes (*R′*) from a new focus. Lead III shows complete A-V heart-block in which each ventricular contraction stimulates the auricles.

sult from the interference within the auricles of the normal and an ectopic impulse. These abnormal and transitional X-waves of Fig. 1 are attributed to the simultaneous spread within the auricles of the normal sinus impulse and an impulse arising in or near the A-V node and due to stimulation by the contracting ventricles. These waves are accounted for in part then in the same manner as are the inverted P′-waves.

Two cases having electrocardiograms similar to those of the present case have been reported, one by Wilson and Robinson,[4] the other by Cohn and Fraser.[5] The latter authors mention similar curves in a case studied by Parkinson. Both of the reported cases have many features in common with the present case. The abnormal auricular waves occur during complete heart block. These waves are practically identical in form and obviously arise in the upper part of the A-V node or in the auriculonodal junction. They are definitely related to ventricular beats, and occur near the end of auricular diastole. They are attributed to mechanical stimulation of the auricles by the contracting ventricles. The ventricular complexes are abnormally broad. In the case of Wilson and Robinson there is spontaneous change in form of the ventricular complexes during complete block. This occurs in the present case also. In these two cases, therefore, it is highly probable that the block is due to a bilateral bundle branch lesion and not to a lesion of the main stem of the His bundle.[1] In the case of Cohn and Fraser, as in the present case, the ventricular complexes are abnormally broad during partial block and differ in form from the complexes of complete block. This suggests that in Cohn and Fraser's case the block was due to a bilateral bundle branch lesion or to a lesion low in the main stem of the bundle and involving one of its branches. In the present case and in that of Wilson and Robinson there was a systolic murmur over the entire precordium. Cohn and Fraser do not describe the physical signs in their patient.

A consideration of the mechanism by which the ventricles stimulate the auricles to contraction during complete A-V heart block must take into account the features mentioned above and also the extreme rarity of the phenomenon. When considered in this light the simple mechanical tug of the contracting ventricles upon the auricles fails as a satisfactory explanation. According to Hering[6] a sudden increase in pressure within a chamber of the heart may cause extrasystoles arising in that chamber. Wiggers[7] and Weiland[8] also regard this as an occasional cause of extrasystoles. The sudden increase in intraauricular pressure which must result from contraction of the ventricles near the end of auricular diastole, with or without mitral regurgitation, might be regarded as an important factor in the production of this rhythm. This explanation, however, is unsatisfactory because it does not explain many features of these records which require explanation. This

rhythm obviously could not be due to retrograde conduction of impulses from ventricles to auricles, in the usual sense, because of the presence of complete heart block.

The stimulation of the auricles by the ventricles during complete heart block finds an explanation which satisfies all of the requirements previously stated when the probable location of the lesion is considered. It is highly probable that the lesion is a bilateral bundle branch block, or that it is very low in the main stem of the bundle. If it is assumed that the bundle above the lesion is functionally intact and susceptible to stimulation by the contracting ventricles, the mechanism of this rhythm is apparent. The contracting ventricles stimulate mechanically the His bundle above the lesion producing the block. The impulse is then transmitted in a retrograde manner through the A-V node to the auricles. It must be considered that the stimulation of the His bundle by the ventricles is mechanical in nature until it is shown that this can occur in any other manner under the same circumstances. It is realized that this explanation lacks the confirmation of autopsy findings and of experimental proof, yet it is in complete accord with the findings in the cases and it is not opposed to any known experimental facts.

SUMMARY

A case is reported in which the auricles respond to stimulation by the ventricles during complete A-V heart block.

This is explained by the mechanical stimulation by the contracting ventricles of the A-V bundle above the lesion producing the block, with retrograde conduction of the impulse to the auricles.

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THE IMPORTANCE AND THE MOST SATISFACTORY CLINICAL METHODS OF ESTIMATING THE MYOCARDIAL RESERVE*

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FROM the standpoint of a physician in any branch of modern medicine, it is impossible to overemphasize the importance of a correct estimation of the myocardial reserve, for it is mainly upon this that satisfactory prognosis and treatment depend.

With the doubt cast upon mechanical defects as the predominating causes of cardiac failure and the introduction of infection as the chief factor, a marked change has occurred in the attitude of physicians towards cardiovascular disease. This article is written with the hope that a more widespread realization of this change in attitude may result in a more accurate clinical estimate of the myocardial reserve.

In reviewing the literature of fifteen or twenty years ago, one cannot but be impressed with the importance then placed upon murmurs, and the intensity or character of the cardiac sounds, in prognosticating the future of a diseased heart. Even today the average student—be he graduate or undergraduate—upon visiting a heart clinic seems most interested in listening to, and in discussing the character and importance of, murmurs. To him that is the *sine qua non* of the diagnosis, prognosis and treatment of heart disease. Certainly murmurs are of importance, and their depreciation is being overdone by many clinicians, but an attempt should be made to seek the middle ground and give them their relative importance as aids in estimating that which, most of all, we wish to know—the myocardial reserve. A systolic murmur at the apex or base, with only questionable cardiac enlargement, may be of great importance in elderly people or during active infections, and practically of no importance in youth, in the absence of infection. In our profession it is almost impossible to generalize, and for the physician the words "always" and "never" should not exist.[1] The history, physical signs and laboratory reports must be interpreted separately for each individual case, and statistics are only of value in giving us suggestive leads toward a more accurate interpretation of the condition of each individual patient.

Before the World War, in the mind of the average physician, any patient complaining of breathlessness, palpitation, fatigue and precordial pain, must needs have disease of the heart, no matter what the physical examination revealed. The "effort syndrome"—whatever its etiology may finally prove to be—has revealed the fallacy of this attitude. Quoting Paul D. White, "breathlessness, tachycardia, cyanosis and heart pain do not alone justify the diagnosis of heart

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disease, as they are found in a variety of conditions, convalescence, fear, fatigue, etc., when the heart is perfectly sound." An accurate, painstaking history is of great importance in the estimation of the myocardial reserve, but most important of all is the interpretation of such a history, with the proper value placed upon each symptom found.

Frequently we hear the terms "poor muscular tone" and "weak heart sounds." If one will think of the number of mediums through which heart sounds must pass, one cannot help being struck with the chance of error in determining the character of the myocardium by the intensity of the heart sounds. The pericardium, the pleura, the lungs, the chest wall, the skin, the stethoscopic bell, the stethoscopic tubing, the physician's auditory apparatus, and finally his interpretation of sound, may, one or all, affect the final interpretation. If we listen to the heart sounds of a thick-chested athlete, and of a thin-chested child with endocarditis and myocarditis, in which shall we find the heart sounds the louder? We certainly must not attempt to interpret the myocardial reserve upon the intensity of the heart sounds, except in the case of sudden changes in those whom we have been able to examine from day to day, and thus are able approximately to estimate the normal intensity of that individual's heart sound.

Again, in the cases of low blood pressure, with or without so-called "weak heart sounds," we have often been at fault in our estimation of the condition of the myocardium. Hundreds of men have been sent to the United States Veterans' Bureau with the diagnosis of "chronic myocarditis" based upon a history of breathlessness and these two physical findings alone.

It must be admitted that we are realizing more and more that the systolic blood pressure alone gives us very little information as to the myocardial reserve. Let me qualify this statement by excepting the knowledge obtained of probable changes in the myocardium in patients with constantly elevated blood pressure; and that obtained by a sudden fall in the sytolic pressure during the course of pneumonia or other acute infections or in long-standing hypertensive cases. In these instances, however, accompanying signs of circulatory failure simplify the proper interpretation of the lowered systolic pressure.

To the average physician an irregular rhythm of the heart means serious disease of the myocardium, and yet the two most frequent arrhythmias, in youth at least, namely sinus arrhythmia and multiple premature contractions or so-called extrasystoles, are of almost no diagnostic or prognostic import, from the standpoint of the myocardial reserve.

So far this paper has been destructive in its comments. If the above facts be admitted, one's faith in those signs which have been taught in medical schools and emphasized by the best recognized of the older writers as indicative of poor myocardial reserve has been questioned. How then is one to estimate whether or not the heart

will stand additional burdens placed upon it, or explain the cause of various symptoms of which patients complain?

As I have already indicated, the answer to these questions should be based upon a proper interpretation of the history as well as the results of the physical examination, and the findings with certain instruments of precision, namely, the x-ray and the electrocardiogram. Let us inquire diligently for evidence of infection, past or present, such as acute rheumatic fever, chorea, scarlet fever, typhoid fever, syphilis and chronic foci of infections, which may have damaged the circulatory system, for a familial tendency toward diseases of the circulation, and for a positive history of the various indiscretions of life which we believe tend toward early vascular changes are of importance. All this may seem obvious but is too often omitted in the rush to listen for murmurs. Mackenzie[2] says, "it is one of the curious matters in medicine that the commonplace and self-evident are the things which are the most frequently ignored. I am repeatedly told by experienced physicians that the views here expressed are 'common knowledge' and that everyone knows all about them. But if justification were needed for insisting upon this line of observation, we have but to observe how the profession employs this common knowledge." Barringer[3] believes that "the best way to judge of an organ's capacity is to set it doing its own particular work and base our judgment upon the result of such experiments." This sounds simple, but we find it to be just as difficult in relation to the heart as the attempts to estimate the ability of kidneys to do their work. The circulation is a complicated mechanism and the myocardial reserve of the heart is only one factor; hence the failure, thus far, in the development of a satisfactory "exercise test." A recent careful résumé has emphasized the fact that no *one* satisfactory "exercise test" has yet been devised and generally accepted by the profession. This, however, should not detract from the definite clinical value of observing a patient's reaction to effort, so long as proper weight is placed upon A routine determination of the vital capacity of the lungs offers much it as one of various evidences as to the state of the myocardial reserve. promise in this connection.

Pregnancy, the greatest of all physical strains placed upon the myocardial reserve, with the exception of that caused by a lack of the proper blood supply to the myocardium, comes readily to mind in any consideration of the subject of cardiac response to effort. Time does not permit of a detailed discussion, but I must agree with the main conclusions of the Obstetrical Society of Vienna in 1921, which were that in the determination as to whether pregnancy should be interrupted or allowed to go on to termination, we must consider not so much the actual condition of the heart as whether it is performing its function properly. The report empha-

sized the rarity of death from cardiac failure in pregnant and parturient women. Among sixty thousand patients there were but eleven deaths directly attributable to failure of the myocardial reserve.

Briefly it may be said that, in estimating the myocardial reserve, auscultation helps us in the following manner: Diastolic murmurs, if persistent, are practically always due to organic lesions, and are therefore most suggestive of changes in the myocardium. The same may be said for true presystolic murmurs. The presence of a true gallop rhythm, at rest or after moderate effort, also is significant of poor myocardial reserve. The French cardiologists place especial importance upon this auscultatory finding, and confirmatory evidence is rapidly accumulating in this country.

The truest estimate of the extent of the encroachment upon the myocardial reserve is to be gained from a determination of the extent of the enlargement of the heart. This dictum of the modern cardiologist is the one least well understood and least readily accepted, perhaps because of the difficulty of determining the extent of enlargement in borderline cases. Nevertheless, any enlargement of the heart which is demonstrable means damage to a greater or lesser degree to the myocardium and a loss of myocardial reserve. Unfortunately the converse of this statement is not true, because of the imperfections in our present methods of determination of cardiac enlargement. We are not justified in saying there has been no loss of myocardial reserve because we are unable to demonstrate enlargement; although such loss is much less likely under such circumstances. Eyster[4] has much ground for his statement that, "determination of the size and contour of the organ represents the most important single procedure in cardiac diagnosis. If we are in a position to detect with perfect accuracy the departure from the normal in this respect, we could, with our present knowledge of cardiodynamics, determine the presence and estimate the extent of every cardiac abnormality, except those specifically affecting the autonomic and conductive system." We attempt to demonstrate enlargement by inspection and palpation. If, in adults, by either or both methods, we can demonstrate a true systolic heave to the precordium, we are justified in diagnosing at least some loss of myocardial reserve. If, by percussion of the right and left cardiac borders, we can be sure of an increase above the normal for that individual with respect to size, contour of thorax, etc., then we have additional evidence of cardiac enlargement. If, under standard conditions, there be a definite preponderance of either ventricle in the electrocardiogram, or if the fluoroscope, and the orthodiascope, or the six-foot x-ray plate, give evidence of cardiac enlargement, we have even more positive evidence of loss of myocardial reserve. Here again one must almost wholeheartedly agree with Eyster when he claims "the most generally neglected of the aids to (cardiac) diagnosis * * * is the x-ray. * * * We do not hesitate to carry

out a comprehensive x-ray study of gastrointestinal cases, and yet the information generally obtained has usually a less direct bearing upon the underlying pathology than in heart disease."

More and more is the string galvanometer being given its proper evaluation. Certainly electrocardiographic tracings will never take the place of clinical findings, but when interpreted with the proper breadth of vision, they are often invaluable in estimating the myocardial reserve. We must admit them to be the final court of appeal as to arrhythmias, and Levine[5, 6] has shown that during operations the development of alarming cardiac symptoms usually has, as the underlying factor, a sudden change in cardiac rhythm. Certainly the future promises much for electrocardiography as an aid in determining the myocardial reserve as is evidenced by the recent reports of Willius[7] on the high mortality in, and prognostic importance of, all cardiac cases with what he calls "significant T-wave negativity."

SUMMARY

In the attempt to estimate the degree of myocardial reserve reliance cannot be placed upon: (1) *certain subjective symptoms,* without accompanying clinical and laboratory evidence; (2) *low systolic blood pressure* and so-called *"weak heart sounds"; (3) systolic murmurs alone;* (4) *a poor reaction to effort alone,* or (5) *certain arrhythmias,* without other clinical evidence of myocardial changes.

On the other hand in estimating the myocardial reserve the importance should be stressed of: (1) a careful investigation for *infections past and present,* possibly productive of damage to the myocardium; (2) a proper *association* of *subjective symptoms* with definite clinical and laboratory findings of *pathological changes;* (3) a proper interpretation of the so-called *"reaction to effort"; (4)* the need of determining the *time* in the cardiac cycle at which *murmurs occur,* and the *character,* rather than the intensity, of the cardiac sounds, and finally, (5) (aided by careful inspection, palpation, percussion, x-ray findings and conservative electrocardiographic interpretations) a determination of that which means more than any one single observation in estimating the amount of myocardial reserve, namely, *enlargement of the heart.*

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STATISTICAL ASPECTS OF THE PROBLEM OF ORGANIC HEART DISEASE*

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This article, by the distinguished statistician of the Metropolitan Life Insurance Company, is republished through the courtesy of the Editor of the *New York State Journal of Medicine* and is offered here because of its vivid and authoritative presentation of the size and importance of the problem of heart disease.—Editor.

HEART disease in its various manifestations is first in the order of causes of death and, I am inclined to think, the first also in the amount of damage it does through disability and invalidism. The evidence for mortality is much more complete and decisive so that I shall give that first. The annual quota of deaths in the United States is now close to 200,000. If present conditions continue, one in every five of the population living at the age of ten will eventually succumb to organic heart disease. The child at ten years of age is now three times as likely to die eventually from heart disease as from tuberculosis. At the age of thirty-five, the probability of dying eventually from heart disease is, among males, nearly four times that for tuberculosis and, among females, the probability is almost six times that for tuberculosis. A revolutionary change has taken place in the general mortality picture during the last twenty-five years coincident with the development of preventive medicine and the public health movement.

It is also very likely that the preeminence of heart disease as a cause of death will increase rather than decrease as time progresses. The gradual improvement in the death rate for such diseases as tuberculosis, pneumonia, and others that are coming under control, will transfer many additional persons to the later ages in life when heart disease is likely to strike them down. Under conditions of twenty or thirty years ago, many of them would have died in early life from the conditions referred to; today, they survive to middle life only to become victims of heart disease, cancer, apoplexy, or Bright's disease. This is an item which must not be lost sight of, especially in view of the fact that the medical profession is not so well organized to control heart disease as it has been for twenty years organized to combat tuberculosis. It is for this reason that I consider heart disease the outstanding problem in contemporary preventive medicine. Nothing within the province of the physician today

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compares with it. I have in another place estimated that if heart disease could be eradicated as a cause of death, every one in the population would have about two years added to his expectation of life—not a small matter when the economic value of a year of life is considered.

The first question I would like to take up with you is whether the mortality from heart disease is beginning to show any signs of improvement. Between 1910 and 1918, the indications were that the mortality rate was rising slightly. The years 1917 and 1918 showed the maximum death rates of this period. The three following years, 1919, 1920, and 1921, were years of low rates, and there is every indication that this was largely due to the elimination of many persons through the influenza in 1918 who would ordinarily have died of heart disease in subsequent years. Beginning with 1922, the trend has been slightly upward, and there is really no saying what the picture in the immediate future will be like. In the first four months of 1925, the experience among the sixteen million Industrial policyholders of the Metropolitan, which is usually very sensitive as an index of what will be found later in the general population, showed an increase of 4 per cent over the same months of the year before. The facts for the fourteen years beginning with 1911 are shown in Table I.

TABLE I

DEATH RATES PER 100,000 FOR ORGANIC DISEASES OF HEART. EXPERIENCE OF METROPOLITAN LIFE INSURANCE COMPANY, INDUSTRIAL DEPARTMENT (AGES ONE AND OVER) AND EXPANDING U. S. REGISTRATION AREA (ALL AGES)

YEAR	METROPOLITAN LIFE INDUSTRIAL DEPT. AGES ONE AND OVER	EXPANDING U. S. REGISTRATION AREA (ALL AGES)
1924	125.5	*
1923	128.7	157.3
1922	126.7	148.4
1921	117.4	140.9
1920	117.0	141.9
1919	113.9	131.0
1918	141.7	153.3
1917	142.0	153.8
1916	140.2	150.6
1915	136.7	147.6
1914	138.1	142.2
1913	140.6	138.9
1912	143.8	142.8
1911	141.8	141.1

*Not available.

Heart disease is preeminently a condition of the older ages of life; but, it is by no means to be neglected as a cause of death in the early years. In 1924, for example, the Metropolitan Life Insurance Company recorded close to twenty thousand deaths from heart disease among its Industrial policyholders. Of this number, 1,600, or 8 per cent, were of persons under the age of twenty-five, and 3,400, or 17

per cent, were under the age of forty. In other words, one-sixth of the insured who died from heart disease were at their prime. At the younger ages of adult life, heart disease is responsible for as many deaths as are all forms of pneumonia combined. After forty, the rate of mortality rises precipitously, and the priority of heart conditions is then unquestioned. But it should not be forgotten that these deaths of middle-aged men and women, in most instances, involve a loss of many years of productive life to which they would ordinarily have been entitled, and often years of diminished efficiency and

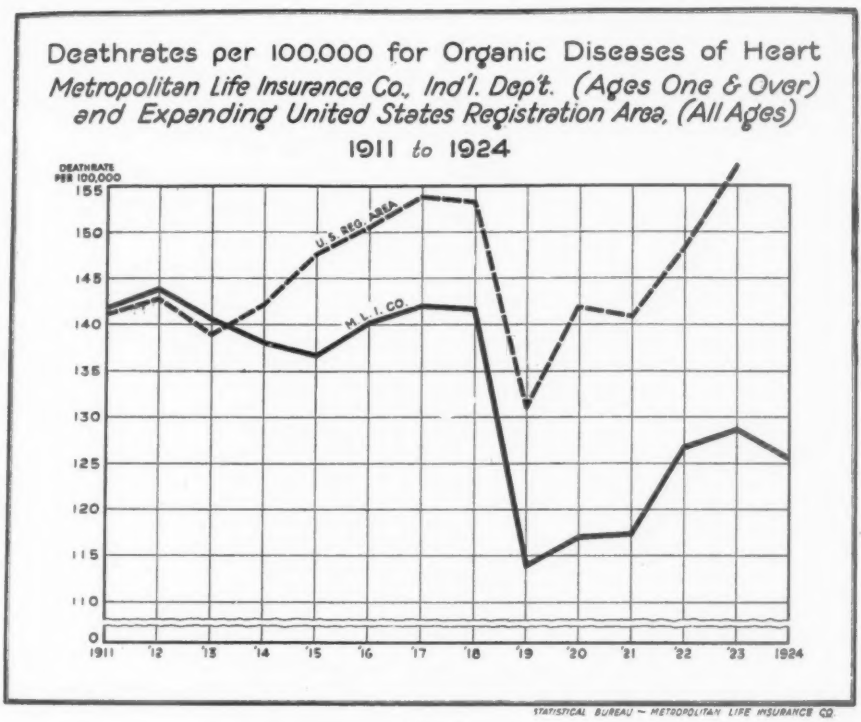

Fig. 1.

even of complete invalidism prior to death. Deaths at these ages also produce in many cases broken families, widows, and orphaned children thrown upon the community for support. The curve of mortality from heart disease by age is a very interesting one, and I present one herewith in contrast with the curve for tuberculosis. The latter disease now shows its maximum at or about twenty-five years of age and then declines with advancing years. But heart disease crosses the tuberculosis curve about the age of forty-five and then mounts to its huge maximum at the oldest ages. The principal damage to the community has, however, been accomplished long before old age is reached. Sixty-eight per cent of all heart disease deaths,

in the experience of the Metropolitan Life Insurance Company, occur before the age of sixty-five.

Among white persons, the death rates are very much the same for the two sexes up to the age of twenty-five. After the age of twenty-five, the death rate for white males is higher than for females and the excess between the sexes becomes greater with advancing age. The rate among colored people is at every age higher than for whites. In fact, during the main age periods of life, the rates for colored people are about twice that for whites at the corresponding ages. It is also noteworthy that at some ages in adult life, the rates for colored females are higher than for colored males. It has been suggested that the higher prevalence of such diseases as malaria, typhoid fever and especially syphilis in the colored race, plays an important

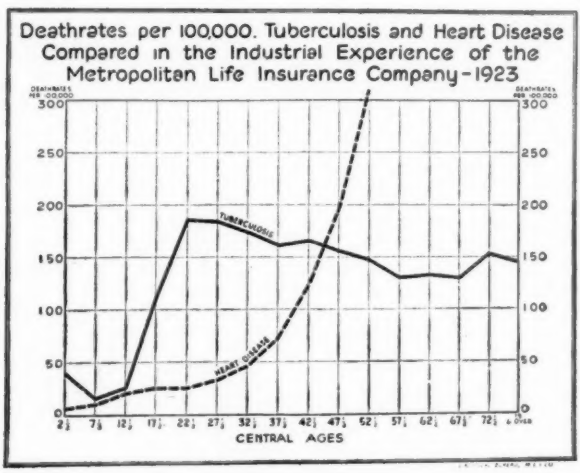

Fig. 2.

part in creating this excess of heart disease among them. But possibly, also, the figures presented are not all that they ought to be, because of the great difficulty in obtaining reliable statements of the causes of death on certificates. We shall always be troubled with our figures for heart disease until there are a larger number of autopsies and the standards of medical practice are generally raised.

I have touched on the principal points in the mortality picture. I wish I could speak with as much assurance on the morbidity aspect of our subject. Unfortunately, that is impossible in the present state of our knowledge. We have made only the merest beginnings in collecting information on the incidence of heart disease in the community. Our very definitions are still vague. I should not have been able to make a presentation today if I had been kept to a literal interpretation of the title of this symposium. For, what is really a cardiac cripple? When does the condition justify such a designation in that

TABLE II

DEATH RATES PER 100,000 FOR ORGANIC DISEASES OF THE HEART. METROPOLITAN LIFE INSURANCE COMPANY, INDUSTRIAL DEPARTMENT, 1923

AGE PERIOD	WHITE		COLORED	
	Males	Females	Males	Females
All ages—one and over	113.6	122.1	190.8	217.4
1 to 4	6.3	6.2	17.1	10.3
5 to 9	10.3	10.8	17.5	10.8
10 to 14	19.8	23.2	28.9	16.0
15 to 19	27.6	23.9	28.4	19.3
20 to 24	23.9	25.2	27.6	38.1
25 to 34	39.6	32.4	68.0	58.7
35 to 44	86.6	70.7	180.3	184.7
45 to 54	253.3	184.9	424.6	470.4
55 to 64	681.3	535.6	831.9	948.8
65 to 74	1719.9	1545.4	1595.6	1641.1
75 and over	4060.8	3524.7	2600.1	3016.5

shadow zone between the functional and organic heart case? Primarily, the difficulty lies in the fact that organic heart impairments ordinarily are not discovered in the early stages, and even then, there is no provision for systematic records of the after-history of the cases. This most valuable information, therefore, lies hidden away either in the memories of the one hundred thousand or more practicing physicians, or in their uncompiled and unanalyzed records. It is only recently that we have made a beginning, through the work of the cardiac clinics, to gather such information as we need on the morbidity of heart disease. It will always be a source of satisfaction to me to have had an opportunity to cooperate with Dr. Afred Cohn in the preparation of the record forms used in these clinics. It is, indeed, fortunate that today we are able to tap this source of information from the unpublished work of Dr. Wyckoff, who has compiled and analyzed the records of a thousand patients in his service. These data are probably the only ones available at the present time, and I wish to acknowledge my great indebtedness to Dr. Wyckoff and his associates who placed this material at my disposal for this occasion.

In a previous paper, I have indicated that the number of persons suffering from definite organic heart disease approximated two per cent of the total population. This is a rough estimate. It is suggested by the findings of the life insurance companies in their routine examination of applicants for insurance; by the findings of the Life Extension Institute in their examinations, and by the results of others who have made physical examinations of large numbers of school children, employees in shops and factories and of other groups. This figure should, of course, be considered only a first approximation and will give place to a more definite one as further work is done on the records. It will serve, however, as our starting point. On this basis, there are well in advance of two million men, women, and children

with organic heart lesions, and if the number of deaths annually from this group of diseases is in effect 200,000, we may infer that the average duration of a case of heart disease is about ten years. This will, of course, vary with the age at which the lesion occurs, with the type of lesion, the care which the individual receives and a host of other factors which bear on the condition. But, this figure of average duration will serve as a beginning to guide our discussion.

What are some of the outstanding findings in the tabulations of Dr. Wyckoff? The thousand cases were distributed etiologically as follows: About one-fourth presented rheumatic heart disease; about

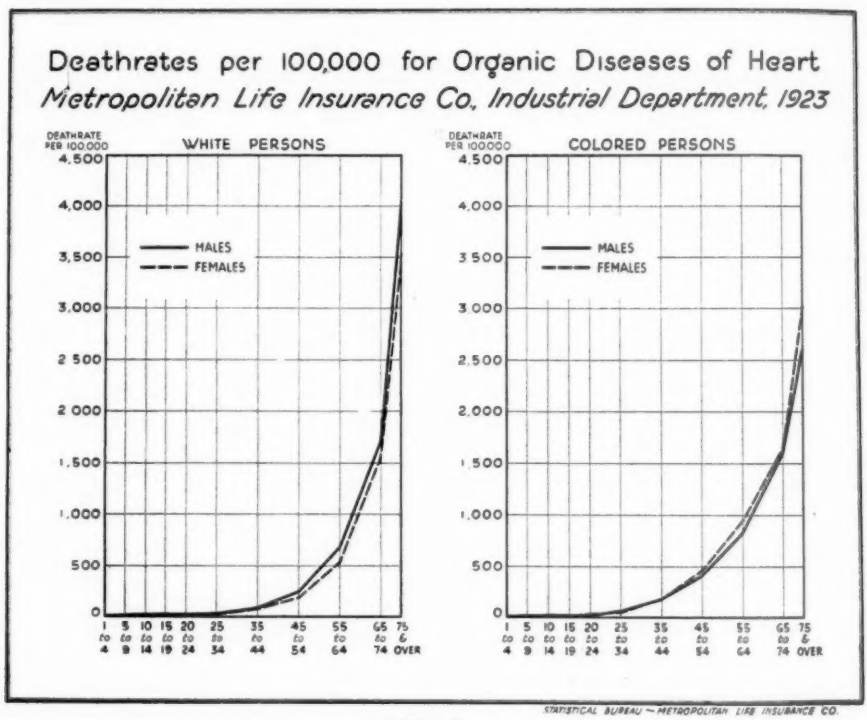

Fig. 3.

two-fifths, arteriosclerotic; about one-tenth, syphilitic, and about another one-tenth, heart disease of unknown origin. The remaining 15 per cent are accounted for by a mixture of odds and ends which are not so easily classified. The cases of rheumatic and unknown etiology are discovered, for the most part, at the earlier ages; the cases of arteriosclerotic etiology appear in the later age groups. The great presumptive importance of rheumatic fever as a causative factor is clearly indicated by the findings of Dr. Wyckoff and his associates. It would be very interesting to find out what the relative duration of these cases of rheumatic origin is in contrast with the duration of cases of degenerative heart disease. The seriousness of syphilis as a

primary causative agent is no surprise. Probably, the figure of ten per cent is a minimal value in view of the difficulty of determining in every instance the presence of the spirochete and the incompleteness of case histories. It is important, however, to observe that this type of heart disease is most prominent in the fifth and sixth decades of life. It is especially among the colored patients that syphilitic heart disease is prominent; in this group, it accounts for about a third of the cases.

An equally valuable classification prepared by Dr. Wyckoff and his associates is that showing the incidence of structural lesions in these 1,000 cases of organic heart disease. They found 88 per cent of the cases showing enlargements of the heart, among whom one in five had enlargement of the heart only. Hypertrophy was, by all odds, the commonest impairment. Mitral insufficiency was diagnosed in approximately half of all the cases. Mitral stenosis was present in 44 per cent of the cases; but, in nearly all of these, mitral regurgitation was also present. Aortic insufficiency was present in about 15 per cent of the cases; aortic stenosis in about three per cent; aortitis in 17 per cent; and aneurysm in a little over one per cent. Table III is taken from Dr. Wyckoff's report, with his permission.

TABLE III

INCIDENCE OF STRUCTURAL LESIONS IN 1,000 CASES OF ORGANIC HEART DISEASE

STRUCTURAL LESION	MALES		FEMALES		BOTH SEXES	
	No.	Per cent	No.	Per cent	No.	Per cent
Enlargement of Heart	542	54.2	341	34.1	884	88.4
Enlargement of Heart, only	137	13.7	66	6.6	203	20.3
Mitral Insufficiency	228	22.8	267	26.7	495	49.5
Mitral Stenosis	216	21.6	227	22.7	443	44.3
Aortic Insufficiency	178	17.8	68	6.8	146	14.6
Aortic Stenosis	23	2.3	6	.6	29	2.9
Aortitis	142	14.2	27	2.7	169	16.9
Aneurysm	11	1.1	1	.1	12	1.2
Mitral Insufficiency and Stenosis	202	20.2	219	21.9	421	42.1
Mitral Insufficiency and Stenosis and Aortic Insufficiency	82	8.2	40	4.0	162	16.2
Mitral Insufficiency and Stenosis and Aortic Insufficiency and Sten.	6	.6	6	.6	12	1.2

This report when it is published should receive the careful attention of all physicians as it is, in fact, the first attempt, to my knowledge, to collect a large body of information of an authentic character in this field of heart disease prevalence. We have, heretofore, had any number of sketchy and loose statements of diagnostic findings. But, this series stands by itself in the authority of the examiners and the fullness of the records. This report should stimulate others in the field to collect similar case records and to tabulate and analyze them along comparable lines. A very great service could be rendered to

this branch of medicine by spreading the use of these forms not only in clinics, but also among physicians in private practice.

I wish now to turn to a third source of information on the prevalence and significance of the heart diseases, namely, the records of the life insurance companies. A number of the larger companies have been for years liberal in their acceptance of certain types of heart cases for substandard insurance. The examinations for insurance are, of course, not made with that same thoroughness which characterizes the work of the cardiac clinics. But the insurance applications do, nevertheless, make possible the classification of the heart findings of large numbers of people with a fair degree of accuracy. The heart defect most frequently found is mitral regurgitation. Hypertrophy of the heart without other heart signs is next in importance. Mitral stenosis, aortic stenosis and aortic insufficiency follow in order of frequency. This order is very much like that of Dr. Wyckoff's material; but, I doubt very much whether the examinations as conducted by insurance medical examiners in the field are of sufficient accuracy and refinement to find all the cases with heart lesions or to diagnose them correctly. Yet, the parallelism is very interesting. The medical directors of the life insurance companies are confronted with the practical problem of evaluating these lives, with placing them in the several risk classes for which mortality rates and premiums have been computed. When insurance is granted, an excellent opportunity is afforded to the medical director of following the subsequent mortality experience on such persons.

A number of companies have, in this manner, collected considerable data on the after mortality of cases with mitral regurgitation which, I believe, will interest you. The experience covers a period of close to twenty-five years and is based on many thousands of persons. Taken altogether, the mortality rate is about two and a half times as high as that which prevails among normal persons accepted for standard insurance, age period being considered. Those cases where the murmur is slight but not transmitted have only a slight excess over normal mortality. The presence of a well marked hypertrophy adds to the hazard, as does also the history of rheumatic fever or other acute inflammatory processes. You will all remember Dr. Mackenzie's very favorable prognosis of these cases. He felt that the insurance companies were losing a great deal of good business in not accepting these cases. But, our experience has shown, I am afraid, that his impressions were not correct, although it may well be that these cases of mitral insufficiency constitute the least impaired of the organic heart cases. In this connection, it is interesting to note that close to half of the deaths which occurred among the mitral insufficiency cases were from organic heart disease of one form or another. Cases of aortic stenosis and of intermittent heart disease gave an experience

very much like that from mitral insufficiency, i.e., double mortality. In a few instances, the insurance companies have accepted risks affected with mitral stenosis, but the experience is uniformly bad, and the present practice is to reject such cases. The available insurance experience is, however, insufficient to give reliable results on the precise effect on longevity of these very serious impairments.

I have mentioned the practice of the insurance companies in this connection with a special point in mind. The medical directors of the insurance companies are keenly interested in the work of your clinics. As physicians they are, of course, very much concerned with the prevention of suffering and the postponement of death. But, as insurance men, they are also concerned with providing an equitable classification of the risks presented to their respective companies. They are all of them very anxious to provide protection for the families of persons affected with impairments; but, it is always necessary that such insurance protection be granted with no unfairness to those who are already insured. For this reason, the medical directors have been compelled to proceed cautiously with the acceptance of risks showing heart impairments. It is quite possible that the new movement for the study of heart disease will provide the very information which will make it possible for the companies to extend their operations to such cases, and to offer insurance to lives which are not now accepted because of the dearth of information on the after mortality of such risks. There are large opportunities for cooperation between your heart clinics on the one hand, and the insurance companies on the other. I believe it would be a very profitable procedure for both groups to get closer together if for no more than to explore the opportunities for further research.

The American Heart Journal

VOL. I ST. LOUIS, MO., FEBRUARY, 1926 NO. 3

Official Organ of the American Heart Association

The American Heart Association

THE American Heart Association, under whose editorial direction this Journal is published, was organized as an outgrowth of a widespread movement for the study of prevention and improved care of heart disease in this country. The organization was completed and incorporated under the laws of the State of New York in May, 1924. Since that time widespread interest has been shown in its activities. Its services have already been extended to heart centers in various parts of the country. It is the earnest desire of the Association that it may rapidly extend its work to all parts of the country.

As a result of the activities of the fight against tuberculosis, it has been realized that while little has been accomplished in recent years to prevent the spread of the disease or to offer specific treatment for its cure, much has been done to provide a field less fertile for the growth of those organisms causing it. A large part of this results from the awakening of public interest and understanding of the ordinary laws of personal and public hygiene. It becomes more and more apparent that only an awakened public opinion will bring about similar results in heart disease.

The American Heart Association therefore seeks the active cooperation of all public and private agencies and individuals who can be of assistance in the solution of any angle of the vast problem. The public must assist in three fields of activity; first, by supporting the work through its active interest; second, by carrying out individually and collectively those principles which have been shown to lessen the incidence of heart disease, also in expecting of public health officials that the carrying out of these principles be made possible for all classes of people; and third, by affording through financial aid facilities for the education and care of those who are temporarily or permanently incapacitated with heart disease.

Physicians must point the way at first in their respective communities toward the correct solution of the problems concerned, and, recognizing the limited knowledge of heart disease, must carry on the work, adding to that knowledge. But it is the public which must make demands for this knowledge in order that humanity may receive from the medical, scientific and educational professions better methods for the prevention of heart disease, and improved ways for the care of those with damaged hearts.

Meeting of American Heart Association

The second annual scientific meeting of the American Heart Association will be held in Dallas, Texas, on April 20, 1926. This date is during the week of the meeting of the American Medical Association. Notice of time and place of the meeting will be given later.

Department of Reviews and Abstracts

Collective Review

THE SURGICAL TREATMENT OF ANGINA PECTORIS

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INTRODUCTION

THE recent introduction of a surgical operation to relieve the pain of angina pectoris has been followed by reports from numerous workers describing patients who have responded more or less satisfactorily to the procedure. The operation proposed originally in 1920 by Jonnesco[1] was to sever the left cervical sympathetic cord and to remove the three ganglia. However, these reports have brought out views as to what operation should be done on the sympathetic nerves and as to the methods of operation which are at variance with the description by Jonnesco. These different operations which have been proposed naturally depend on an explanation of the phenomena which form the symptom-complex of angina pectoris. They have revealed that as yet the exact paths of conduction of afferent and efferent impulses through the sympathetic plexus to the heart are not clearly understood. While the manifestations of angina pectoris have been clearly recognized since the original description of the condition by Heberden[2] as yet no explanation has been uniformly accepted. The operations which have been performed have, in many instances, brought out additional information and, according to the interpretation by the observer, seem to offer one or another explanation of the condition.

Since this divergence of opinion has arisen, it becomes apparent that further work is necessary to clear up the question of the nature and cause of angina pectoris and on that will depend the operation that should be performed for its relief. It is also apparent from the case reports in many instances that an exact diagnosis cannot be made, so that there must exist some confusion as to what constitutes the symptom complex. This review is presented to direct attention to this confusion of ideas and to the different explanations that have been offered as to the cause of angina pectoris. With this in mind it is hoped that the condition will eventually be so clearly defined that patients may be properly selected for an operation which will relieve them of their pain without harm to their subsequent course.

THE NATURE OF ANGINA PECTORIS

Angina pectoris, first described by Heberden[2] as a sensation of strangling and anxiety, is characterized always by pain of a definite kind with a typical localization, coming on in attacks which recur with a fair uniformity. In the interval between attacks the patient is relatively free of symptoms. No physical signs other than the expression of pain have been found constantly with the condition, nor has there been found any structural change in the heart or blood vessels which occur constantly in the many cases that have been examined post-mortem. The condition must be classified, therefore, as a functional disorder of the circulation. That pain simulating angina pectoris may arise from structural change in the heart and vessels is well known.

The clinical picture of angina pectoris does not often vary much from the excellent description of its course by Wenckebach,[3] and Allbutt.[4] Briefly these authors describe the sensation as a pressure, a heavy weight or constriction of the chest which is spoken of by the patient with a grave face and awed respect. Sooner or later, sometimes from the first attack, there is a definite sensation of pain. They may be tearing pains, or burning, as hot fire; one will never hear the expression of "stabbing pain" which is so typical in heart neuroses and in some kinds of irregularities. These sensations are described by the neurotic patient, with dramatic emphasis, and sweeping gesture, pointing with the finger to that part of the chest where he thinks his heart is to be found.

The pain is nearly always somewhere on the sternum at a higher or lower level, mostly at the manubrium. It may be at the level of the third or the fourth rib where the patient will place three or four fingers. In like cases the patient may move his pointing finger across the chest at the angle of Louis, speaking of a tearing pain. It may radiate as in neuralgia or may start and remain in the form of definitely circumscribed paresthesias. The radiation is to the back, opposite the painful area in front; it runs up to the left shoulder, down to the inner side of the arm and may go down as far as the wrist or the little finger. It may ascend to the left or to both sides of the neck, to the angle of the jaw, or even as high as the scalp. These sensations in the neck are often felt as a kind of strangulation. The radiation to the neck and head are important symptoms to be kept in mind when the operative procedure is outlined and in the observation of patients after operation. The pain may be left-sided, right-sided or on both sides.

Typical segmental areas of hyperesthesia or hyperalgesia or of hypoalgesia and anesthesia may be accompanied by tenderness on compression of skin, muscles and nerves; they correspond with the posterior roots of the lower cervical and first one or two thoracic segments. (Head[6] and Danielopolu.[7])

One more important peculiarity of the pain is its absence at rest

while the patient keeps quiet bodily and mentally. At first the pain appears on severe exertion only; later the least effort causes the pain so as to make life quite unbearable. Three other conditions may be themselves the cause of the onset of pain in the great majority of cases: first, a full stomach; second, excitement and nervousness; and third, chilling. These facts indicate that everything which stimulates the heart action, increases its output, and raises the blood pressure, causes the pain.

As soon as these causal influences are removed, the anginal pain will disappear. These peculiarities give anginal attacks a typical aspect; the more so as the pain is of a most excruciating character and so dominates its victim that he is forced to stand still immediately. The patient does not stop still in the street because he finds it convenient or because he knows it will do him good, but because he must. Dyspnea does not belong to angina pectoris. The patient may tell about loss of breath; he may describe curious sensations of suspended respiration and a feeling of constriction which makes respiration nearly impossible. During the attack there are no signs of forced or difficult breathing. With the subsidence of the attack these respiratory symptoms immediately disappear and a deep sigh is the signal of the patient's release and the onset of normal breathing.

The action of nitrites administered during the course of an attack shows a characteristic response by the patient. This action of the drug is of considerable diagnostic importance in differentiating other forms of heart pain, particularly coronary thrombosis.

The clinical findings on physical examination are not characteristic or constant. Percussion and x-ray may or may not show a wide aorta and an enlarged heart. One may find sclerotic or weakened arterial walls; a high or a low blood pressure; angina may occur in the body of athletic build as well as in that of a weakling. The physical signs are of considerable importance in the selection of patients for surgical operation, a point to be discussed later.

Wenckebach states that a certain number of patients in private practice may live for a number of years if they will avoid bodily exertion, mental excitement and full meals and "some of our sweet poisons." A small percentage of patients may die in one of the first attacks. The pain may disappear for a short time; nearly always it comes back. The symptoms of angina may disappear and be replaced by symptoms of heart failure. Other exceptional and complicating conditions may develop or lead to a change of symptoms.

Allbutt[4] sets forth three postulates concerning the condition which he considers of prime importance: (1) that in the very large majority of cases angina pectoris is due to disease of the thoracic aorta and especially to its outer investment wherein lie the sensory end organs that regulate the blood pressures; (2) that death in angina pectoris is due

ordinarily to vagus inhibition, i.e., to the shock of the pain; (3) that the coronary arteries and the myocardium have nothing to do with the pain of angina, but much to do with its mortality. In younger persons in whom the myocardium is healthy, the heart usually survives the inhibition so that in time angina, as often in the case of syphilitic aortitis, for example, may be a terrible, if not a mortal disease.

In persons under middle age angina is, for the most part, of syphilitic origin. Cases occur now and then in the course of other acute infections, more frequently perhaps in influenza, next in rheumatic fever and malaria.

Other points to be considered in the diagnosis of the condition are:

(1) A regular heart action which is usually slow.

(2) No signs of palpitation or other sensations from the heart itself.

(3) A countenance which is normal or pale, never flushed.

(4) The awe-stricken attitude during the attack.

(5) A blood pressure which is often elevated, rarely lowered, and usually unchanged from the pressure present between attacks.

(6) The great predominance of the condition in males.

DIFFERENTIAL DIAGNOSIS

There are two conditions occurring with symptoms resembling the common chronic form of angina pectoris which must be differentiated. These two forms are pseudoangina, a form of cardiac neurosis with pain as a prominent feature, and coronary thrombosis. Pseudoangina is most frequent, by far, among women. The pain is correctly defined as precordial and varies from a tiresome submammary ache to a paroxysm of distressing pain, often striking very sharply along the intercostal and left anterior axillary line, and even down the arm. The patient is not awe-stricken, but is restless and emotional; the face is flushed. These patients are, for the most part, young. The heart rate is rapid, often with irregularities. Palpitation and conscious throbbing are constant symptoms. There may be present other signs of emotional disturbances elsewhere in the body.

Coronary thrombosis or embolism occurs usually under much the same circumstances as angina pectoris, and at the time of onset of the attack the differential diagnosis may be difficult. It has been pointed out[3] that until the attack comes on the patient may have been in seemingly good health; perhaps there had been already some premonitory signs of beginning heart disease; perhaps the patient just felt his heart and found himself short of breath on strong exertion. Suddenly and without obvious cause, often while sleeping, a most severe pain in the chest, increasing into a horrible paroxysm, sets in. The patient is unable to move about, sometimes collapses from weakness or he may become unconscious and die. The attack, if not fatal, does not pass off, but lasts for hours, sometimes days, without any real amelioration. Nitrites are

of no benefit. Only repeated injections of morphine are able to help the pain. The pulse often becomes rapid and frequently shows change in rhythm.[5, 8] The blood pressure always falls and a fever and leucocytosis[9] occurs. Of particular importance are certain abnormalities shown in the electrocardiogram. They indicate or strongly suggest that infarction of the heart has taken place. When at last the patient recovers, he never returns to his previous state of good health. It has been pointed out by Levine[5] that he may survive the attack and eventually be able to carry on a useful life, or may seem to have recovered, but sooner or later signs of heart failure develop. It has been emphasized by all workers that with the appearance of heart failure the cardiac pain disappears. Instances have been shown where under successful treatment for the heart failure, the pain may return. The usual course is that the heart failure once established leads eventually to death.

Reflex pain from other parts of the body occasionally simulate angina pectoris, though in these cases the differentiation offers no particular difficulties. Pain arising in the gall bladder and kidneys, or gastric crises of tabes dorsalis may be mentioned.

CASES TO BE SELECTED AS SUITABLE FOR OPERATION

Conservative opinion points out the dangers of subjecting any or all patients suffering from angina pectoris to operation and warns against the enthusiasm for a procedure newly developed for the relief of a chronic disabling condition. MacKenzie[10] bitterly denounces the operative procedure and points out the danger to be risked. His chief objections are based on the present lack of knowledge of cardiac pain and of cardiac nerve supply. He also points out that the requirements of work for the heart with the nerves cut are not known. Since they are not known, it may be that an additional burden is placed on the heart which is already embarrassed. He bases this objection on his opinion that angina pectoris is associated with, and results from, myocardial damage and heart failure. Since this is a disputed point, his objection may or may not be valid. He believes the cause of death in the operated cases to be due to ventricular fibrillation. Furthermore, he points out that in relieving the pain of angina pectoris by operation, a danger signal which safeguards the patient against excessive exertion may be removed to the detriment of his future course. Even, in fact, death may be hastened.

Levine and Newton,[8] however, believe that with careful selection of patients the operation may actually prolong life and at the same time relieve the patient of a most distressing feature of the disease. They point out that the repeated attacks of angina tend to provoke other attacks with probable injury to the heart. They state that two important points must be borne in mind: (1) that the operation should be

reasonably certain of producing no damage to the patient whether or not any benefit is derived. (2) It should be the aim to select those patients who may be expected to live long enough to benefit from any satisfactory results that might follow the operation. Younger people, therefore, who, under proper rest and care are free more or less of attacks and who without rest are unable to carry on their daily occupation, even though this be restricted and adjusted, would seem to offer the best opportunity.

These authors have described criteria for selecting cases. If a patient shows evidence of myocardial damage sufficient to produce congestive heart failure, it is unlikely that he will be a good surgical risk and it should not be forgotten that anginal attacks may spontaneously cease as congestive failure sets in. Furthermore, the coexistence of valvular disease must be duly considered, for although angina pectoris is uncommon in mitral stenosis, it is not so rare in the syphilitic type of aortic insufficiency,—a progressive disease causing myocardial changes from which patients may die suddenly, even in the absence of failure. The signs which indicate a favorable condition of the myocardium are a knowledge that the attacks come only on effort; that they are of short duration and that there has not been much shortness of breath. There should be no suspicion that cardiac infarction is taking place at the present time or has occurred in the past. The vital capacity of the lungs should not be very much depressed and the electrocardiogram should show essentially normal complexes. It would be best if all the T-waves were upright and the QRS complexes sharp and of fair amplitude.

On examination the quality of the heart sounds should be good, there should be no gallop rhythm or pulsus alternans. The rhythm should be normal. The actual size of the heart is not of great importance, as most patients with angina show only slight or moderate cardiac enlargement. Marked enlargement would indicate that grave myocarditis exists and should be a danger signal. The presence or absence of systolic murmurs has no practical significance; neither has the question of precordial hyperesthesia. The exact level of the systolic blood pressure is also of minor importance, for it may vary from normal to an extremely high reading. One must bear in mind, however, that a very low pressure of 90-110 mm. may be the result of a previous infarction of the heart and demands investigation from this point of view.

It has been pointed out by others[11] that the prognosis of syphilitic disease of the aorta without aortic valve insufficiency is favorably influenced by antisyphilitic treatment. It would seem from this opinion and those of other workers that surgical interference for angina pectoris in syphilitic aortitis should be undertaken only after a thorough trial of medical treatment.

EXPLANATION OF THE ORIGIN OF THE PAIN IN ANGINA PECTORIS

For a long time there have been three explanations of the origin of the pain in angina pectoris, each explanation being advanced as a theory substantiated by experimental work and clinical evidence. Since the information recently obtained by a section of various sympathetic nerves and paths of conduction for efferent and afferent impulses to and from the heart, other theories have been advanced to explain the condition. Briefly, these theories may be set down as follows:

1. Heart muscle failure. This theory has found its most ardent enthusiast in MacKenzie.[10, 12] He maintains that narrowing of some part of the coronary vessels with degenerative changes in the heart muscle may be found at autopsy.

2. Coronary artery disease, coronary thrombosis or embolism may produce attacks simulating angina pectoris. The differentiating points have been presented above. Coronary sclerosis or narrowing was first described as a cause of angina pectoris by Parry and Jenner.[13] Since, on further study, this has not been found constantly at autopsy in hearts of patients dying of angina pectoris and in those hearts which showed marked sclerosis with no history of angina during life, this idea has been given up.

Paroxysmal vasomotor spasm of the coronary arteries has been also held to be a cause of angina pectoris. This explanation has been likened to the pain in intermittent claudication of voluntary muscles. Whether or not this may be true remains to be proved. Such experimental evidence as may be set forth points against such a possible explanation. In intermittent claudication the pain is present only in the muscle involved, and does not radiate. Also the pain in claudication occurs not only during the stage of diminished or absent blood supply, but also during the period of relaxation when the blood begins to flow and distends the vessels. In this connection it is worthy of note that the existence of vasomotor nerves to the heart has not been accepted as proved. It has been shown that epinephrin applied directly to the vessel causes constriction, but that when administered through the general circulation, there is a dilatation of coronary vessels, probably as a possible result of the increased blood pressure in the peripheral vessels.

3. Disease of the base and ascending arch of the aorta. This explanation of the nature of angina pectoris has been set forth most convincingly by Allbutt.[14] In brief he maintains that due to changes in the vessel wall at the base of the aorta there is irritation of the nerve endings of the sympathetic cardiac plexus and that with rise of intra-aortic pressure there results pain. This theory has found many workers, notably Wenckenbach,[3] who accept it as the explanation of the pain in angina pectoris.

4. Reid[15] has offered an explanation of the pain in angina pectoris which depends for its main principle upon facts that are in keeping

with the ideas expressed by Allbutt. He has emphasized the circulatory changes which take place as a result of exercise, pointing out evidence to show that there is a peripheral vasodilatation, especially in the voluntary muscles involved in the exercise, and that there is probably at the same time a splanchnic vasoconstriction. The dilatation of the peripheral stream bed causes a more rapid drainage of the blood away from the heart and lessens the rise of pressure in the first part of the aorta, and also the height of that which must be achieved in the left ventricle before the aortic valve can be forced open. This dilatation of peripheral vessels takes place as a result of reflex stimulation through the various sympathetic paths. When the peripheral arteries fail to dilate or are unable to dilate as a result of reflex stimulation from the heart, there is a rise of intraaortic pressure producing pain.

The sequence of events in angina pectoris may well be a failure of the reflex dilatation of the peripheral vessels, which leads to a sudden rise in the pressure of the first part of the aorta and of the cavity of the left ventricle. This heightened pressure in turn irritates the local nerve end plates which respond by pain referred to the arm, shoulder, etc.

5. Danielopolu[7] offers an explanation of the pain in angina pectoris which is not related to any of the others set forth above. It would appear that the mechanism may be somewhat comparable to vasomotor spasm of the coronary vessels, though this factor is not mentioned by him. He considers advisable to attribute the cause as due to an inadequate coronary circulation in a physiological sense. This inadequate circulation may occur when the arteries are healthy, but it is more likely when they are in a state of sclerosis. The explanation appears to be as follows: the anginal attack arises from a disturbance of the balance between the work of the myocardium and its blood supply through the coronary arteries. It is conceivable that even healthy coronary arteries, forced by an excessive demand, may be unable to respond sufficiently to provide an adequate myocardial blood supply, and consequently it is not out of the question that such a disturbance of balance may occur in perfectly healthy hearts. Moreover, this suggestion explains those cases of coronary disease without anginal symptoms where the slowness of growth of obstruction in the coronary artery has permitted the development of anastomotic channels and the provision of a good blood supply. When an anginal attack occurs in healthy hearts after overexertion, there is present a condition of coronary insufficiency without coronary disease. In the case of extensive coronary obstruction without anginal attacks there is present coronary disease without coronary insufficiency. Furthermore, in cases of angina without coronary disease but with basal aortitis only, the coronary orifice is often involved. While the hypothesis of coronary inflammation only explains a single class of cases, that of coronary insufficiency is applicable to all.

He believes that the starting point of the angina is in the myocardium, more precisely in the left side of the heart, and suggests that the process is analogous to that which is concerned in the fatigue or voluntary muscle. The inadequacy of coronary blood supply arising from one or another contingency leads to myocardial poisoning and cardiac fatigue. From this there results sensory symptoms and also motor phenomena which may end in the heart ceasing to beat. The former are due to the stimulation of sensory nerve endings in the myocardium and later to a change in the motor endings.

OPERATIVE PROCEDURES

The operation for the relief of pain in angina pectoris was first performed in 1916 by Jonnesco[1, 16] after he had carried it out many times for the relief of other conditions such as epilepsy, migraine, trifacial neuralgia, thyroid disease, etc. His results in angina pectoris were most gratifying and led others to attempt the same operation. His original operation consists in removing the middle and lower cervical and upper sympathetic ganglia on the left side. He recommends that the ganglia on the right side should be resected if there is no relief from pain.

Resection of all three cervical sympathetic ganglia and the upper thoracic ganglion has been reported by Brüning[17] and Kappis.[18]

Resection of the main cervical sympathetic trunk and the superior cardiac branch of the superior cervical ganglion reported by cardiac branch of the superior cervical ganglion has been reported by Coffey and Brown.[19] In a later report[20] these authors modify their views and recommend the removal of the superior cervical sympathetic ganglion only.

Reid and Eckstein[21] observed marked sensory disturbances after removal of the superior ganglion and suggest that the operation be modified to consist of an excision of the cervical sympathetic trunk, together with the middle and inferior cervical ganglia, the first thoracic ganglion and the division of the superior cardiac branch of the superior cervical ganglion.

Eppinger and Hofer,[22] working independently in Wenckebach's clinic, have devised an operation which consists in severing the depressor nerve. This nerve, described by Cyon, arises by two branches, one from the vagus and the second from the superior laryngeal nerve. In man it does not always exist independently as a separate trunk in all instances. When found it seems larger on the left side. Section of this nerve relieves in part the pain of angina pectoris.

Borchard[23] resected the depressor nerve as described by Hofer and also removed the superior ganglion of the cervical sympathetic.

Danielopolu and Hristide[24] anesthetized the second and third left spinal nerves with relief of pain in a case of angina pectoris. The distribution of pain corresponded to the area supplied by these nerves.

Danielopolu[25] therefore recommends resection of the posterior roots of the left dorsal nerve corresponding to the area of pain as a surgical treatment of angina pectoris. He draws attention[7] to the fact that from all the various operations performed on the cervical sympathetic system good results have been reported. There seems no doubt that the pathway of pain has been interrupted in the performance of many of these operations and that patients have been improved.

Holmes and Ranson[26] report one case operated on and advance an opinion to explain the relief of pain. They state that no sensory pathways for the heart in man are found in the cervical sympathetic trunk going to the superior ganglion or in the superior cardiac nerve which descends from the ganglion. They advance the hypothesis that the superior cardiac nerve contains vasoconstrictor fibers for the aorta and coronary arteries and that section prevents spasmodic vasoconstriction. This in turn stops the paroxysmal pain of angina pectoris, but is without effect on pain caused entirely by structural changes.

Penfield[27] reports two cases that were relieved of their pain after resection of part of the sympathetic cervical nerves. However, after operation other pains and sensory phenomena appeared. These pains simulated in part the original pain of angina. In order to explain these phenomena he calls attention to reflex pain arising as an autonomic reflex, so-called by Langley and Anderson. He states that the success in these operations depends not upon interrupting the direct afferent path from cardiac plexus to central nervous system as has been assumed, but from the interruption of these autonomic reflexes. By stimulation of sympathetic axones in the cardiac plexus the motor cells in the intact sympathetic ganglia would be excited, causing localized sweating, arterial spasm and some increase in blood pressure.

It becomes apparent from this survey of the various operations devised and the opinions expressed in connection with them that what is needed is a more thorough knowledge of the anatomical or physiological relationships of the nerves to the area of the heart and vessels. Relief apparently is obtained in many different ways when it seems that no path has been disturbed. This brings up the opinion expressed by Coffey and Brown[20] that at operation as little should be done as is consistent with fulfilling the original purpose of the operation, i.e., to relieve pain. In the more extensive types it may be that serious damage is done to structures essential to heart function. This warning has been sounded by many authors.

It seems apparent that, with the many operations devised, sooner or later more knowledge will come about for these relationships in human beings. This can be a most fortunate result of the efforts of these pioneer workers. The condition cannot be recognized at the autopsy table, and like many other physiological phenomena, must be worked out on the living human being.

EXPECTED RESULTS FROM OPERATION

That there is relief of pain from operation is clearly recognized. This relief comes from almost any type which has been worked out. Only a few of the workers claim to have completely relieved the symptoms and several state that, while the original pain has disappeared, others have taken its place which caused the patient almost, if not as much, distress.

The general mortality from the operation has varied. From a survey of the literature it is difficult to form any accurate opinion. It can be stated that the immediate mortality from the operation itself is extremely low. The ultimate mortality is complicated by the fact that these patients are in danger of sudden death at any time from various causes. Among these may be mentioned thrombosis of the coronary system, vagus inhibition, ventricular fibrillation and left ventricular muscular failure. These patients, therefore, may die from one of these causes whether or not they have been operated upon, so that for this reason it is difficult to ascertain the direct relationship between the operation and death. The distinct warning is issued by several workers that there should be no interference with the stellate ganglion, as this may involve serious disturbance in the heart action. One result of operation which is uniformly observed is the so-called Horner's syndrome. This is characterized by enophthalmos, myosis, pseudoweakness of palpebral muscles, narrowing of the palpebral aperture and absence of sweating and flushing on the affected side. This picture appears immediately after operation, particularly when the superior ganglion is removed. It is usually a permanent change, but instances are recorded where the signs disappeared.

Lewit[28] expresses his opinion that the heart muscle suffers as a result of the operation and that this effect is not desirable. He thinks that by relieving the pain the automatic brake which has kept the patient from overexerting his heart will result in disaster. No effect on the thyroid gland has been noted as the result of operation.

Schiff and Heinrich[29] extirpated the cervical ganglia in dogs and subsequently examined the thyroid gland. They found no changes which they interpret as signifying that no real benefit is to be expected from the operation in exophthalmic goiter.

One further effect of the operation has been referred to by MacKenzie[10] He maintains that when the pain is relieved, a most important danger signal for the guidance of the patient is removed. This objection is well founded and must be considered in every patient when the postoperative care is being considered.

Ormos[30] describes pigmentation and degeneration of cells in the cervical ganglia of three fatal cases of angina pectoris which he examined. There was no definite sclerosis or other changes in the coronary arteries.

No other pathological changes in the nerves or ganglia have been described by other workers.

CASE REPORTS

In this review no attempt has been made to summarize individually the various cases which have been reported. In addition to cases noted in the various references given, there are numerous instances of other patients who have been operated on for this condition. They are referred to as bibliographical references by the author:[31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41] This no doubt is only a partial list as numerous cases are known not to have been reported.

SUMMARY

In summary the opinions which have been expressed as to the value of operation in angina pectoris lead one to conclude that it is a procedure of great value for the relief of this distressing condition when all other measures have been tried without success and when the operation is done on patients who are carefully selected. In such instances the operative mortality is low and the results permit the patient to lead a reasonably comfortable life for a greater or lesser period of time. When this much can be done there must be a distinct value to a surgical operation. It must be understood by the patient that it is only a palliative remedy and is in no way an attempt to cure the original condition. More light in the future on the various processes involved in the transmission of pain will assist in indicating the type of operation to be carried out.

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Selected Abstracts

Pichon, E.: Le Rhumatisme Cardiaque Evolutif et son Traitement.. (Progressive Cardiac Rheumatism and Its Treatment.) L. Arnette, Paris, 1924.

In his monograph, Pichon presents the available evidence of the existence of a chronic, progressive form of rheumatic infection of the heart, based on personal observations and on cases previously reported. As generally described in the text-books, when the rheumatic virus invades the heart, the disease runs a more or less acute course, and after the inflammation has subsided there remain healed lesions consisting of valvular defects, pericardial adhesions or myocardial scarring. The injection may be eradicated after initial attack, the patient suffering only from the mechanical effects of cicatrized lesions, but more frequently the infection recurs. Between attacks the joints and heart are believed to be free of active inflammation. The author points out that there exists in addition to the simple acute and recurrent rheumatic infections of the heart a large group of cases in which the inflammation persists in chronic form over a period of months or years. The author applies to these cases the designation "progressive cardiac rheumatism" or "chronic rheumatic carditis." It may be noted that Bard as early as 1892 called attention to the frequency of subacute and chronic inflammatory lesions in the hearts of cardiopaths of long standing dying of cardiac insufficiency ("asystole inflammatoire").

Many cases of well-established valvular disease are really instances in point and in these the manifestations of persisting rheumatic inflammation may easily be overlooked. Endocardium, pericardium or myocardium may be involved either individually or in combination. Of the cardiac phenomena encountered in chronic rheumatic carditis, frank myocardial insufficiency is the least frequent except as a terminal event. The commonest manifestations of cardiac failure seen in these cases are enlargement of the liver and oliguria. Diarrhea, sometimes postprandial, is occasionally observed. Precordial pain is frequent and may occur independently of pericarditis. Pain of myocarditic origin is described as cramp-like in character and attended by oppression and a sense of fullness in the precordium. It is uninfluenced by bodily movements and respiration and is sometimes relieved by percussion and massage.

Dyspnea is an equivocal symptom because it may be due to cardiac disease without active inflammation. When it occurs it is sometimes associated with precordial

pain and oppression. According to Gallavardin, the patient may suffer from paroxysms of pulmonary edema. Palpitation is a frequent symptom and there may be forcible and conspicuous pulsation at the base of the neck even in the absence of aortic insufficiency.

The heart is generally enlarged, but the enlargement may vary from time to time, increasing during a febrile attack and diminishing after salicylate therapy. Muffling of the heart sounds is considered an important sign for the diagnosis of inflammation in the heart valves, but may also be due to pericardial effusion or myocardial dyspragia. The murmurs heard in chronic rheumatic carditis may be classified as follows: (1) Murmurs due to cicatrized endocardial lesions, e.g., mitral stenosis. Such murmurs may be muffled during the course of the chronic rheumatic infection. (2) Murmurs due to functional insufficiency caused by ventricular dilatation. (3) Murmurs developing during the course of the disease due either to inflammation in the valve sphincters (localized myocarditis) or in the valves themselvse (valvulitis).

The presence of pericarditis signifies active infection. Small effusions may occur but massive ones are rare. Disturbances in the rate and rhythm of the heart, e.g., tachycardia, bradycardia, extrasystoles and auricular fibrillation, are not infrequently encountered. (The author does not sufficiently emphasize the importance of electrocardiographic alterations as a diagnostic aid in determining the presence and persistence of the rheumatic infection in the heart.)

Small elevations of temperature persisting over a long period are frequently observed. On the other hand, the absence of fever by no means signifies the absence of infection. (No mention is made of leucocyte counts.) There may be chills or chilliness and acid sweats similar to those encountered during the acute stadium of the disease. Sore throat, purpura and epistaxis may occur.

The articular phenomena vary from fleeting arthralgias to frank arthritis with redness, swelling and pain in the affected joints. The joint symptoms are often inconspicuous in children, but not infrequently in adults as well. In certain cases the same joint becomes inflamed during each recrudescence. The persistence of tenderness in one of the joints long after the signs of acute inflammation have subsided is regarded as an important symptom. Muscular pains and neuralgias also occur (cervico-sciatic syndrome described by Nobecourt and Peyre: Bull. et mem. soc. med. d. hop. d. Par., Feb. 25, 1916).

The disease may be progressive from the start or secondarily so. In the former, the disease becomes chronic after the initial attack (which may go unrecognized). In the latter, the subject of a cicatrized cardiopathy due to antecedent rheumatic disease develops a recurrence of the infection which in turn becomes chronic.

In the differential diagnosis, the important conditions to be distinguished are (1) the postrheumatic cicatricial cardiopathies, especially valvular defects, with or without myocardial insufficiency but uncomplicated by existing rheumatic infection and (2) the various forms of nonrheumatic infections of the heart valves, particularly subacute bacterial endocarditis.

The author believes the salicylates to be specific for rheumatic infections. Once the diagnosis of progressive rheumatic heart disease has been made, salicylate therapy, preferably sodium salicylate, shou'd be continued as long as there are signs of rheumatic infection, however slight, and if necessary for many months. Treatment should be begun with large doses and continued with repeated courses of small doses. No toxic effects on the myocardium were observed by the author when the drug was given in therapeutic amounts. The method of choice for administering salicylates is by mouth, but in the presence of gastric intolerance the drug may be given intravenously or per rectum.

Swift, H. F.: Rheumatic Fever. Am. Jour. Med. Sci., 1925, clxx, 631.

At a time when interest in this most important disease is being revived, this article summarizes the present status of our knowledge concerning its cause and course. Himself an active student of the disease, the author brings together in review opinions of his own and of other workers. He draws a most interesting picture in pointing out the factors in common between syphilis, tuberculosis and rheumatic infection. The resemblance between tuberculosis and rheumatic infection is especially close when one considers the relationship of heredity and environment to the etiology and to a consideration of the contagious nature of the two diseases. Also in studying the various types of manifestations in patients of different ages it is apparent that severe general acute manifestations are more frequent in the young and that in succeeding decades of life there is a transition to more localized lesions.

He states that the etiologic agent of rheumatic fever has not been demonstrated conclusively nor has it been possible to reproduce in animals the characteristic clinical and histo-pathological picture of the disease. In this connection he points out that the endocardial vegetations and myocardial lesions so characteristic of the disease known as subacute infective endocarditis are also seen in animals properly inoculated with Streptococcus viridans; nor is it possible to demonstrate by complement fixation, precipitant reactions or other biological phenomenon that a streptococcus is responsible. One hears constantly reference to the "toxin" or "poison" of rheumatic fever when it is known that a streptococcus does not form an exotoxin.

The pathologic changes are described in detail as they occur throughout the body, principally the subcutaneous nodules in the tissues about the joints and in the heart muscle, endocardium and pericardium. He describes these changes under two heads; proliferative and exudative. At the bedside the only visible proliferative lesion is the subcutaneous nodule, but the interpretation of many symptoms and signs as well as of the peculiar course of the disease is made simple by the knowledge that comparable changes are occurring in important organs.

The exudative response, on the other hand, is more evident in the swelling, pain and tenderness seen in acute rheumatic polyarthritis. Probably the most characteristic feature of this disease is the disappearance of exudation and the symptoms depending upon it following the exhibition of sufficient doses of certain drugs. It is well established nevertheless that subcutaneous nodules may appear continuously in a patient who is receiving maximum doses of salicylates.

He places importance on the study of the heart's action with the electrocardiograph as furnishing evidence of functional disturbance. Transitory or a continued delay in conduction, or changes in the ventricular complexes occur in 90 per cent of the cases and indicate that probably the heart is infected in every patient suffering from rheumatic fever.

The weight curve of patients is a serviceable guide in helping to determine whether or not the patient is succeeding in overcoming his infection. In the author's experience the maintenance of nutrition is one of the most important therapeutic measures. He describes the value of other therapeutic agents, particularly salicylates. The value is largely to render the patint asymptomatic. This serves to relieve the hyperpyrexia, often the severe general intoxication, reduces the heart rate, and because of the reduced rate the amount of injury to the valve cusps is lessened.

The most important phase of treatment of rheumatic fever is the length of time it is necessary to keep the patient quiet. The author thinks this should be as long as signs of infection persist. In a chart showing the duration of stay of 72 patients in the Rockefeller Hospital, the average time is about 100 to 110 days, varying from extremes of 50 to 260.

Wright, Hedley D.: Bacteriology of Subacute Infective Endocarditis. Jour. Path. and Bacteriol., 1925, xxviii, 541.

I. Technic of Blood Culture.—Media which contains trypsin to 5 per cent, or sodium citrate not to exceed 0.2 per cent are slightly superior to those which do not. Addition of lactic acid has not produced any improvement in the media. Liquid and solid media are equally suitable. There is no advantage to be obtained by the use of anaerobic methods. The media employed will permit the growth of organisms if they are present even in such small numbers as 1 to 2 per c.c. If such media are used failure to isolate a streptococcus from the blood indicates the absence of such organisms from the specimen examined.

It was found that the fibrinated blood revealed on culture only one-tenth of the number of colonies which grew from an equal amount of citrated blood. Delay in inoculation of the media with infected blood does not lead to destruction of the organisms in the blood if it is kept at room temperature. If, however, undiluted blood were exposed to incubator temperature, clotting being prevented by the aid of sodium citrate or heparin, it became sterile in the course of twenty-four hours. During the first five hours the reduction in the number of bacilli was small. On the other hand, when trypsin is added to blood in a concentration of 20 per cent and the blood is incubated, not only is destruction of the streptococci prevented, but growth actually occurs. Blood treated in this way is therefore especially suitable for transmission to a distance for examination.

II. The Delay in the Appearance of Growth of Streptococci.—In blood cultures from cases of subacute endocarditis the delay is largely due to prolongation of the latent period. This phenomenon of the delay appears to depend upon the certain peculiarity of the organisms rather than upon inhibitory factors in the blood.

III. The Significance of the Organisms Isolated from the Heart Blood after Death.—Terminal invasion of the blood stream by pyogenic organisms in cases of subacute infective endocarditis has been demonstrated. Streptococci are shown to be the commonest organisms found in the heart blood after death from causes other than subacute infective endocarditis. Agonal and postmortem cultures are therefore unreliable. The isolation of a streptococcus of hemolytic or nonhemolytic type from the cadaver is far from conclusive evidence that that organism is etiologically related to the disease from which the patient suffered.

IV. The Frequency and Peculiarities of the Septicemia.—In 12 of 19 cases of subacute infective endocarditis, positive blood cultures of nonhemolytic streptococcus were obtained. The positive cases were always positive and the negative cases always negative. There is a general tendency for the number to increase in the later stages of the disease, but the increase is not regular and there may be long periods when it remains stationary. It has not been possible to draw any conclusions from the number of the organisms as to the length of time the patient will survive. The temperature of the patient at the time of taking the blood has had no effect on the results. Cases yielding negative cultures have done so even if the temperature was elevated. In cases with septicemia the number of organisms found has not been greater when the temperature was high and has in fact sometimes been smaller than when it was low. These observations then lend no support to the view that the septicemia is intermittent in character. On the other hand, they suggest that even one negative culture is significant in the majority of cases. They do appear to demonstrate that in certain cases of the disease no bacteria can be cultivated from the blood.

The blood of septicemia cases contains antibodies and is actively bactericidal. The septicemia is considered to be a secondary phenomenon, being due to escape of the organisms from the lesions in the valves.

V. Microscopical Examination of the Vegetations.—Where the blood culture has been positive, bacilli are readily demonstrated in the valve lesion. In the non-bacteremic cases bacteria are present in the vegetations in the characteristic position, but the organisms seem to remain *in situ*, they being dead or they may be prevented from reaching the blood stream by the physical barrier of the thrombus material separating them from it. At the same time they are protected from the phagocytic cells by the same barrier. Consequently, a process of removal by solution or autolysis is necessary and is apparently a very tardy one. Whether the process does ever go on to complete removal and subsequent healing by fibrosis still remains doubtful, but it would appear to be quite possible, though apparently rare. Bacteria may be found in the blood of nonseptiecmia cases which show evidence of undergoing disintegration.

The nature of these organisms has not been definitely demonstrated by cultural methods, but they appear microscopically to be streptococci.

Telia, L.: Considérations sur le Syndrome de L'Angine de Poitrine dans la Stenose mitrale. (Observations upon the Syndrome of Angina Pectoris in Mitral Stenosis.) Arch. de Mal. du Coeur, August, 1925, xviii, p. 531.

A case is reported in detail of a girl, aged seventeen, with mitral stenosis who for more than a year before she died had attacks of radiating precordial pain and oppression closely resembling angina pectoris. At autopsy the aorta and coronary arteries were found free of disease, both grossly and microscopically. From a study of his own cases and that of others, the author concludes that the attacks of severe precordial pain which sometimes occur in mitral stenosis are due either to compression of the left coronary artery between the left auricle and the pulmonary artery or to compression of the aorta and the aortic plexus by the dilated left auricle. Venesection (300-500 c.c.) is recommended for the relief of the auricular congestion.

Mentl, S.: La Valeur Diagnostique des Changements Prononces dans la Position et dans le Volume de L'Oreillette Gauche du Coeur. (The Diagnostic Value of the Pronounced Changes in the Position and Volume of the Left Auricle.) Arch. de Mal du Coeur, February, 1925, xviii, 76.

In mitral disease, the greatly dilated left auricle is often found displaced to the right because of the dilatation and rotation of the heart. This occurs especially in children and in adults with flattened chests. If the mitral defect is accompanied by aortic insufficiency, the hypertrophied left ventricle also facilitates rotation. Clinically the displacement of the dilated auricle may be diagnosed by dullness to the right of the spine in the lower half of the interscapulo-vertebral space, the extraordinary propagation of the heart sounds to the back, especially towards the spine or even to the right of the latter, and the occasional occurrence of auricular pain to the right of the spine posteriorly. Valuab'e confirmatory evidence can be obtained by observing the position of the barium-filled esophagus in the x-ray. The esophagus follows the contour of the auricle and when the dilated auricle is displaced to the right, the esophagus is found to the right and the rear.

Krumbhaar, E. B., and Crowell, C.: Spontaneous Rupture of the Heart. Amer. Jour. Med. Sci., clxx, 828.

The authors have collected from the literature reports of 632 cases of this accident and to this add 22 of their own cases. This accident occurs, on analysis of this series, most frequently in males and in the latter part of life. The average age is well in the sixties. The terminal symptoms are usually sudden in all cases.

The mechanism of death may be extensive hemorrhage from cerebral anemia, but more probably from cardiac arrest due to some disturbance of cardiac mechanism. The left ventricle is the usual seat of the tear. Many underlying causes are mentioned, the usual being coronary thrombosis or coronary disease with myocardial lesions. This lesion is usually an acute infarct.

MacLachlan, W. W. G.: The Relation of Morphology to the Prognosis of Aortic Syphilis. Am. Jour. Med. Sci., clxx, 856.

The author points out that syphilitic aortitis is most commonly localized in the thoracic portion of the aorta. When the aortic ring or valves are involved in the process spreading from the aorta there is aortic regurgitation with heart failure. This type of lesion is associated with a grave prognosis.

When the aortic ring is not involved in the syphilitic change, the prognosis is much better, and the case may show definite improvement by treatment.

Crawford, J. Hamilton, and McIntosh, J. F.: The Use of Urea as a Diuretic in Advanced Heart Failure. Arch. Int. Med., 1925, xxxvi, 530.

Urea was given in doses of from 30 to 60 grams a day to eight patients with advanced heart failure in whom the treatment of the heart per se did not suffice to remove edema or to maintain the patient free from symptoms. In these cases a marked improvement in the clinical condition of the patient took place which could be assigned to the action of urea. The treatment succeeded in maintaining an adequate urine output and also in removing edema. As soon as the administration of urea was stopped, the urinary output immediately fails, and the clinical condition becomes worse. When treatment was resumed an improvement again took place. They have studied some of the cases for months and have found that a particular dose will give the same daily urine output with only slight variations throughout the period investigated. In these cases it has seemed that the maintenance of an adequate water excretion has been instrumental not only in preventing symptoms but also in avoiding a relapse.

Urea is rapidly absorbed and an increase immediately occurs in the height of the urea in the blood. The mean level of the urea in the blood is dependent on the dosage, and the relationship between them is fairly constant in any particular case. The amount of urea excreted depends on the blood urea, so that with constant urea administration a state of equilibrium is reached between the intake and output. During urea diuresis the excretion of water runs parallel with that of urea, so that the urine volume reflects the concentration of urea in the blood. Undoubtedly the explanation of the diuresis is that the excess of urea circulating in the blood is excreted by the kidney, and during the process carries with it a considerable amount of water.

Gordon, Burgess: The Value of Venesection in the Treatment of the Decompensated Heart. Amer. Jour. Med. Sci., 1925, clxx.

The author studied 31 patients who were suffering from heart failure. Of these it was necessary to perform the operation of venesection in 12 patients. By x-ray examination it was determined that the extremely dilated heart was reduced to one only moderately dilated. It seems to the author that the venesection improves the action of the decompensated heart and temporarily, at least, removes signs and symptoms of failure.

White, Paul D.: The Use of Drugs in Heart Disease. Boston Med. and Surg. Jour., 1925, excii, 283.

The author discusses in order digitalis, quinidin, adrenalin, caffein, as well as other diuretic drugs, diuretin, theocin, calomel and calcium chloride; camphor,

strychnine, the nitrites and bacteriocidal agents such as mercurochrome. He concludes with the statement that in the practice of any drug therapy one must not lose sight of other treatment. Rest and diet may accomplish more than digitalis or quinidin and the results of such measures must not be credited to drugs.

Adrenalin is classed as an emergency cardiac stimulant to be used in cases of cardiac standstill, especially during surgical operations or anesthesia.

Caffein is a circulatory stimulant with a vaso-motor effect which is of help in shock, as a respiratory stimulant in Cheyne Stokes respiration and as a diuretic.

Camphor and strychnine are stated to be without any effect on the circulatory system.

The nitrites are described as useful in angina pectoris, the author preferring nitroglycerin.

The very important subject of the use of mercurochrome and gentian-violet as bactericidal agents in acute endocarditis is discussed as disappointing. Also the action of salicylates in acute rheumatic fever with heart disease is dismissed without adequate discussion; while opinion differs as to the efficacy of these agents, there are many who feel that their use is indicated at times.

It is disappointing to note that the author does not mention morphine and its derivatives as a drug which is useful in the treatment of heart disease. Next to digitalis morphine may be used more than any other drug for its beneficial effect in providing the much needed rest for the patient's mind, body and heart.

Digitalein and quinidin are discussed at length and in a most conservative and clear manner. He lists three indications for the use of digitalis: (1) evident congestive failure (2) permanent auricular fibrillation (3) as a therapeutic test. He then discusses the three usual effects of digitalis on the heart: (1) heart block (2) reduction of rate in normal rhythm and (3) the direct stimulation of the myocardium. He states there are two complications as associations of congestive failure that occasion a poor response to digitalis, fever and hyperthyroidism. The various methods of using the drug, the choice of preparation, standardization, etc., are presented and discussed. The use of quinidin in auricular fibrillation is discussed. The author feels that in spite of reported unfavorable results at times, that it is a drug with a distinct place in a list of useful remedies when properly used in selected cases.

Bendove, R. A.: The Circulatory Changes in Artificial Pneumothorax. Am. Review Tuberculosis, 1925, xii, 107.

The report includes studies on 14 patients treated by artificial pneumothorax; two cases of spontaneous pneumothorax and nine cases that had had previously x-ray observations. Eight cases showed an increase of systolic pressure from 10 to 40 mm. of Hg., a diastolic increase from 10 to 25 mm. lasting about one hour after inflation; 4 cases showed no change at all and 2 cases had a slight fall. The 8 cases with increased pressure deve'oped enlargement of the heart about ten days after the beginning of treatment; one case with a fall in pressure also showed an increase in the transverse diameter of the heart. In 4 cases in which the pneumothorax therapy was about to be completed and the treated lung was expanding and functioning almost to its physiologic limit, the heart previously enlarged became reduced in size.

Accentuation of the pulmonary second sound was found in those cases which developed cardiac enlargement and a rise in blood pressure. In the cases which showed a definite increase in the transverse diameter in the heart but no rise in blood pressure after inflation the pulmonary sound was not accentuated.

Marked compensatory emphysema was noticed in all patients who had had an increase in the size of the heart, and a rise in blood pressure. The emphysema

was most pronounced when the treated lung was almost totally compressed because of high intrapleural tension or by fluid. Those cases in which the intrapleural pressures were almost negative and the uninvolved portion of the treated lung expanded and contracted with each respiratory phase developed emphysema also in the functioning portion of the lung.

Adamson, J. D.: Basal Blood Pressure. Canadian Med. Assn. Jour., 1925, xv, 1112.

Blood pressure is analogous to metabolic rate in its variations; therefore no one should give an opinion as to the significance of blood pressure findings unless they are taken under conditions in which factors that may cause physiologic variations have been eliminated.

The effect of digestion is probably inappreciable one to two hours after a meal. The effect of ordinary exertion seems to pass off after lying down for fifteen minutes. The recumbent supine position as a standard eliminates variations due to posture. The effect of mental and psychical stimulation can only be overcome by repeating the procedure so frequently that it becomes commonplace to the patient. It may be assumed that the basal level is reached when several successive readings show no further fall.

Apfelbach, Carl W.: The Effects of Diphtheria Toxin on the Myocardium of Guinea Pigs. Jour. Infect. Dis., 1925, xxxvii, 443.

This paper is concerned with the changes which occur in the myocardium of 27 guinea pigs inoculated with diphtheria toxin. In 18 the injection was made subcutaneously, using a dosage from 0.5 c.c. of 1:1000 dilution to 0.8 c.c. of 1:40 dilution. The hearts were removed at a time preceding the expected death of the animal, fixed and sectioned. Eight animals were injected directly into the heart muscle.

The outstanding changes are retrogressive. Fatty changes are the outstanding alternation. The fat is commonly found in patches in the outer and inner thirds of the walls of the ventricles in the form of round minute granules in the muscle bundles, more numerous near the nuclei. Cloudy swelling was found in most of the hearts. Definite evidence of necrosis was found only in 5 of 18 animals. Interstitial change in the sense that it is used in the description of diphtheritic myocarditis was observed in two instances.

Infiltration of leucocytes with degenerative changes occur, but in the myocardium of guinea pigs degenerative proliferation and exudation were not commonly observed together in the same lesions.

Jonnesco, T.: L'Etat Functionnel du Coeur après L'Extirpations du Sympathigues Cervico-Thoracique. (The Functional State of the Heart after Cervico-Thoracic Sympathectomy.) Bull. de l'Acad. de Med., Oct. 27, 1925, xciv, 919.

During the course of an operation on an incompletely anesthetized subject, Jonnesco isolated a nerve which could be followed from the superior laryngeal to the aorta. Stimulation of the cephalic end produced a fall in the blood pressure, whereas stimulation of the aortic end was without effect. This is the first time, according to the author, that a nerve corresponding both in position and function to the depressor nerve in animals has been demonstrated in man. It is of interest to note that stimulation of this nerve produced no pain.

In a number of patients in whom unilateral or bilateral cervico-thoracic sympathectomy had been performed for angina pectoris and other conditions, there was no reduction in the blood pressure, the pulse varied within normal limits and there was no change in the electrocardiogram or in the roentgenogram of the heart.

The author concludes that the cardio-accelerator nerves, which are sectioned in this operation, are not of vital importance and advocates the removal of the stellate ganglion along with the cervical sympathetic ganglia and nerves for the relief of angina pectoris.

Horsley, J. S., Jr.: The Healing of Arteries after Different Methods of Ligation. Jour. Am. Med. Assn., 1925, lxxxv, 1208.

Single and multiple ligations of the arteries of dogs with and without section of the artery were made using catgut, silk and linen sutures. The brachial, femoral and carotid arteries were used. The end results following ligation were about the same for all materials used as sutures, although the author states that catgut is absorbed more quickly and should not be used for the ligation of large arteries.

The changes occurring in the vessel walls and surrounding tissue after ligation are described as the formation of an internal callus with healing. Small blood vessels found in the callus may penetrate through and restore the vessel lumen. When the vessel is cut or crushed by a ligature the ends retract and the narrow permanent cicatrix closes the ends of the vessel. The larger the artery and the higher the arterial pressure the greater is the tendency to a reestablishment of the arterial lumen.

If an artery is severed between ligatures the stump immediately retracts and the process of healing is somewhat similar to the healing of the artery after ligation in continuity. However, after section the intima of the stump obtains an additional amount of nutrition from the surrounding raw tissue and the exposed stumps are soon filled with a plug of granulation tissue. This plugging of the exposed ends of the diseased artery and the original retractions of the stumps from one to several centimeters from each other make the chance of reestablishment of the arterial channel apparently impossible.

The author recommends two or more ligatures and severing the artery between the two distal ligatures. The proximal ligature takes up the impulse of the heart beat and permits the tissue within the grasp of the distal ligature to heal at rest.

Bickel, G., and Frommel, E.: Recherches Expérimentales sur la Pathogénie des Arythmies, Arythmies Extrasystoleques, Arythmie Complète par Fibrillation Ventriculaire Consécutives à L'Injection Intraveineuse d'Extrait Thyroidien à Doses Massives. (Experimental Studies on the Pathogenesis of the Arhythmias, etc.) Arch. de Mal. du Coeur, July, 1925, xviii, 461.

By injecting large doses of fresh thyroid extract of sheep into rabbits, the author succeeded in producing a variety of arhythmias, viz., extrasystoles, complete heart block, paroxysmal tachycardia and auricular and ventricular fibrillation.

Leriche, R.: Recherches Expérimentales sur L'angine de Poitrine. (Experimental Studies on Angina Pectoris.) Presse Med., Oct. 14, 1925, xxxiii, 1361.

In a man who had never suffered from angina pectoris, electrical stimulation of the left stellate ganglion provoked intense pain in the precordial area and the first two or three left intercostal spaces. The examination was made under local anesthesia, but the anesthetic had not been given deeply enough to penetrate the sympathetic ganglia. The pain was accompanied by a sense of oppression, but there was no dyspnea apart from that which was caused by the pain. The injection of novocaine into the ganglion abolished the pain at once.

In several other cases, electrical stimulation of the upper pole of the first thoracic ganglion provoked sharp pain in the arm on the corresponding side, whereas stimulation of the lower half of the same ganglion gave rise to precordial

pain. Similar but less intense symptoms resulted from pinching the ganglion. Stimulation of the last two cervical rami communicantes caused pain in the corresponding arm and shoulder.

A woman on whom a sympathectomy was being performed for the relief of angina pectoris developed an attack during the operation before the rami communicantes had been cut. Injection of novocaine into the left inferior cervical ganglion instantly stopped the attack. These observations furnish valuable information concerning the mechanism of the attacks in angina pectoris.

Danielopolu, D., and Proca, G. G.: Role des Nerfs du Coeur dans la Production des Contractions Ectopiques. I. Contractions Ectopiques Provoquées par la Compression Oculaire et la Compression du Vague. (Role of the Cardiac Nerves in the Production of Ectopic Contractions. I. Ectopic Contractions Provoked by Ocular and Vagal Compression.) Arch. de Mal. du Coeur, October, 1925, xviii, 625.

Compression of the eyeballs or the vagi in normal subjects produces slowing of the pulse, changes in the P-wave and at times isolated nodal and idioventricular contractions. The changes are more marked after binocular than monocular compression, more after ocular than vagal compression and more after compression of the right vagus than of the left. Right and left ocular compression give about equal results. Ectopic contractions are more easily provoked in individuals suffering from myocardial disease, and in general the effects of ocular and vagal compression are more conspicuous than in healthy subjects. To a lesser extent this is true of individuals with disorders of the vegetative nervous system, e.g., hyperthyroidism. In an epileptic in whom the cervical sympathetic and stellate ganglia of both sides had been removed, slowing of the pulse was more intense and prolonged after ocular compression than after vagal compression, but the latter was followed by a greater number of ectopic contractions than the former. The appearance of ectopic contractions does not therefore depend in these observations in slowing of the rate alone.

Book Reviews

THE PHYSIOLOGY OF EXERCISE.—By James Huff McCurdy, M.D., Director of Physical Education in the International Young Men's Christian Association College. Pp. 242. Lea & Febiger, Philadelphia and New York, 1924.

To the medical man (as well as to those whose primary interests are in the physical training and development of athletes), this book will be of value as a means of reaching a great deal of the work which has been done in establishing standards for normal adolescents and young adults. There are sections on *heart rate* and its variation with age, altitude, and exercise of various types, duration and intensity; on *blood pressure* and the modifications produced by age, temperature, tobacco, posture and exercise; on *respiration*, with discussions of vital capacity, variations due to sex, age, training, exercise; on the *blood* and the changes produced by exercise, altitude and other factors; on *muscle* development and coordination. The book consists in the main of a condensation of the literature, but to this Dr. McCurdy had added a considerable amount of original material, the result of 25 years of his own studies and of those conducted by students under his direction. As one reads this volume, one wishes that there could have been less condensation, more critical analysis and a more amplified discussion of many of the facts presented. One has the feeling that he must read the original papers if he is to obtain a true evaluation of the facts which Dr. McCurdy quotes. That the literature has not been completely covered can be seen from such omissions as that of the work of Henderson on acapnia; of Addis, and of Symonds on blood pressure; and of Peabody on vital capacity. The physician is indebted to Dr. McCurdy for making easily available many of the physiological standards which he must daily use as a control in studying pathological conditions.

T. S. H.

Erratum

In the article by Dr. Riecker, in the December issue of the journal, the last word in the last line, Micrococcus, on page 194, should have been omitted.